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HEMORRHAGIC CYSTITIS AND TUBERCULOSIS OF THE PROSTATE*

Case Report

BY O. DRAPER PHELPS, M.D.†

A man of forty-eight, a wire worker, entered the hospital complaining of hematuria which had existed since the day previous.

Family and past history were essentially negative.

Five months ago, he noticed some pain along the urethra, a frequency and discomfort when voiding, particularly in the erect position.

His blood pressure was 130/100. Heart normal. Blood picture normal. Bleeding and clotting time normal. Prostate not enlarged. Bleeding was persistent and excessive. He was cystoscoped with unsatisfactory results on account of the free bleeding. He was drained by catheter and there was no let-up in his bleeding.

On admission, red count was 4,300,000; white count was 15,000. Hemoglobin 70 per cent.

The man was operated upon with the expectation of finding a tumor of the bladder. The operation was done under spinal anesthesia. The bladder was full of clots, no tumor, calculus or areas of definite ulceration were found. The bladder wall seemed to ooze blood from nearly every portion, especially where touched by sponge or instrument. The bladder was closed around a large Freyer tube.

At the time of operation, his red count was 2,380,000; white count was 10,750 and hemoglobin 50 per cent.

Postoperative diagnosis was hemorrhagic cystitis.

His after-treatment consisted of intravenous saline, glucose and a transfusion, together with a nutritious diet. His convalescence was slow, but gradual.

On his discharge, the red count was 5,700,000 and hemoglobin 70 per cent.

In the London *Lancet*, Fullerton, in reviewing 605 cases of hematuria, found the bladder as the source in 470 and of these cystitis and tumor were the most common causes. Bladder injuries and calculi were next in importance.

Podvinec and Pollak in a German periodical mention a vesical purpura as the source of bleeding, and Lichtenstein in another article from German literature mentions varicosity as a cause.

The other case is that of a man fifty-eight who entered the hospital on account of a pain in his back and difficulty in locomotion. He was admitted to the Medical Service.

He was examined carefully. There was noted a slight irregularity in the prostate, a nodule in the left lobe. There were no urinary symptoms, no residual. Cystoscopy was negative and the stains and guinea-pig examination of bladder and both kidney urines were negative.

The spine showed a destructive lesion involving the upper half of second lumbar. Impression was that of tumor, probably malignant.

The diagnosis was probable malignancy of prostate.

This man was operated upon for confirmation of diagnosis.

Suprapubic cystotomy was done under ether anesthesia. A portion of the left lobe of the prostate was removed and examined by our pathologist, Dr. Raymond Goodale, by frozen section and no malignancy was found. The wound was closed with drainage. The recovery was uneventful.

On staining and further examination of the portion of prostate removed, our pathologist reported the finding of tuberculosis. The slides were reviewed and confirmed by Dr. Shields Warren and others at a Cancer Clinic which met at our hospital at that time.

We feel that this case may be another one of tuberculosis of the prostate gland.

Before leaving the hospital, an Albee fusion was done to immobilize the diseased vertebrae.

There are comparatively few cases which are considered definitely as tuberculous lesion of the prostate. Usually a primary focus is located in some part of the body. We found no primary focus in this case.

Patch and Foulds in the *British Journal of Urology* in 1931, reported tuberculosis of the prostate in the absence of tuberculosis elsewhere in the genito-urinary tract as extremely rare.

Lowsley and Duff in *Annals of Surgery* in 1930, considered tuberculosis of the prostate as usually secondary.

Barney in 1923, reported a case of tuberculosis of the prostate. The total number of such cases is not large.

Both the patients here reported have obtained a satisfactory restoration to their usual health. The tuberculous prostate case is working and it is a year since his operation. The hemorrhagic cystitis case is working and it is about six months since his operation.

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VESICOINTESTINAL FISTULA*

BY HAROLD L. LELAND, M.D.†

THE chief interest in the case I have to present tonight lies in the chronological development of events, and I will endeavor to present it from that viewpoint.

On August 4 of last summer, Mrs. A. was admitted to the Ward Genito-Urinary Service of the Lowell General Hospital. Her chief complaint was dysuria and great frequency, of three months' duration, with hematuria for the past three days. The only fact of interest in her past history was her story of so-called "bilious attacks" with "much gas", beginning four years ago and recurring at intervals to date, until overshadowed by the urinary symptoms. These attacks were accompanied by slight nausea but never by vomiting. She was constipated and had required a laxative for several years.

Physical examination on admission showed a small emaciated woman, appearing much older than her fifty-one years. The only abnormality to be found was a slight tenderness in the left iliac region; no masses could be palpated.

The laboratory reported a normal blood count, a blood urea of 20 mgm., and an alkaline urine of a very foul odor, having a specific gravity of 1.022, a trace of albumen, and a very heavy sediment filled with pus and some blood.

Cystoscopic examination on the day of admission revealed a bladder with a very offensive urine, so turbid that repeated washing would not permit identification of any landmarks. There was a large, gray, ovoid shadow lying in the base of the bladder, coinciding with the x-ray picture.

A suprapubic cystotomy was performed under novocain infiltration anesthesia. The stone was removed and the bladder closed with a double layer of sutures, being drained with an indwelling urethral catheter.

Her postoperative recovery was excellent. Her temperature never rose above 99°F. She had no discomfort, although her urine remained very foul.

Her wound appeared to be healing by first intention until the ninth day, when it suddenly broke open and discharged a large amount of semi-formed feces and urine.

My immediate thought was that, in closing the bladder under the somewhat restricted field of local anesthesia, a stitch had caught a coil of gut and had finally sloughed through into the bladder. I labored under this disquieting illusion for a week, when the intern came to me and said: "Doctor, I have just talked with Mrs. A.'s husband. He tells me that she was passing fecal matter through her urethra for a month before she entered the hospital, but was too embarrassed to tell us about it."

My morale immediately rose 100 per cent.

In addition he said: "Here are two stones which she passed last month." One, measuring about two cm. in length, contained as a nucleus a small piece of wood, resembling a piece of broken toothpick; the other, a small piece of string.

I accused Mrs. A. of inserting the toothpick into her urethra. This she indignantly, and I think truthfully, denied. We then theorized that the toothpick had entered the bladder from the intestine, causing the fistulous tract.

Her further convalescence was uneventful but slow.

Cystoscopy on the twenty-first postoperative day was unsuccessful inasmuch as the bladder could not be cleared of its fecal content. She refused a gastrointestinal x-ray examination and was discharged on the twenty-fourth postoperative day with the suprapubic sinus well healed, but the bladder still contaminated with feces.

SECOND ADMISSION

Mrs. A. returned September 1 as an out-patient.

Cystoscopic examination was again unsatisfactory because of the fecal content of the bladder.

A cystogram at this time showed a practically normal bladder contour.

THIRD ADMISSION

On September 15, Mrs. A. again returned as an out-patient.

This time cystoscopy showed a marked improvement. The mucosa revealed a chronic cystitis with a small area low down on the posterior wall which was depressed, irregular and seemed to be the site of the fistula. It did not appear malignant. Attempts to pass catheters and filiforms into this tract were unsuccessful. Both ureters showed a cloudy urinary efflux. They were catheterized in spite of the infected condition of the bladder. Specimens from both kidneys showed pus, bacteria, and a positive culture for colon bacillus. The phthalein test was normal.

Bilateral pyelograms with sodium iodide 12.5 per cent showed very slight changes which we interpret as being due to infection.

FOURTH ADMISSION

October 4, two months after her original entry, Mrs. A. was again admitted to the hospital. This time she complained primarily of obstinate constipation, with colicky abdominal pains, distention and vomiting. This condition had been developing gradually during the past month.

Physical examination showed a horseshoe-shaped abdominal distention, suggestive of a distended colon from obstruction in the sigmoid. No masses could be felt.

She was relieved by repeated high enemata and pitressin.

During this stay in the hospital a quart of dilute methylene blue was given as a high rectal injection. Her bladder was then viewed through the cystoscope for three quarters of an hour without any color being seen. A bladder injection of methylene blue appeared in the feces the next day.

At this time we felt that she was suffering from a partial intestinal obstruction. We were undecided as to whether this obstruction was caused by a new growth, or by adhesions about the fistulous tract, which apparently was high in the colon, the semiformed feces excluding the small gut.

She again refused a gastrointestinal x-ray examination, signed a release and left the hospital against advice on the fifth day.

FIFTH ADMISSION

December 3, four months from the date of her first admission, Mrs. A. again entered the hospital. This time she was admitted to the General Surgical

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Ward Service, with a recurrence of her intestinal obstruction.

Physically she had lost some weight and her general condition was not nearly so good.

The urine still contained a small amount of fecal matter.

Cystoscopic examination again failed to permit the introduction of a catheter or filiform into the fistulous tract. There was nothing in the bladder suggesting malignancy.

A gastrointestinal x-ray, now accepted for the first time, showed a filling defect in the sigmoid which could not be filled by a barium enema.

On the tenth day an exploratory operation was attempted under spinal anesthesia, with the hope of doing a colostomy.

The abdominal cavity contained a small amount of serous fluid. In the pelvis was a hard mass the size of one's fist. The transverse colon dropped down and was adherent in this mass. The sigmoid and a coil of small gut were likewise involved. The bladder was adherent low down on its posterior wall to the transverse colon.

The bladder was easily separated. A small adhesion, no larger than a lead pencil, contained the communicating tract with the transverse colon. This was severed and the bladder and colon closed in double layers.

Attempts were then made to bring the sigmoid up for a colostomy. This was impossible because of the adhesions and the great fragility of the tissues, which tore and spilled feces into the abdominal cavity.

Finally the peritoneum was brought down to the sigmoid, low in the pelvis. The abdominal wall was partially closed with profuse drainage.

The patient did not rally. She died the next day, undoubtedly of surgical shock.

Autopsy could not be obtained, but a pathological examination of tissue removed from the sigmoid at operation showed a frankly malignant adenocarcinoma, grade II.

To summarize: We had a simple vesical calculus, with an incomplete and therefore misleading history; a fistulous tract between the transverse colon and bladder with subsequent intestinal obstruction from an adenocarcinoma of the sigmoid.

The unsettled question is the relation of the toothpick in the first calculus to the conditions found. Did it cause the fistula, or was degeneration of the carcinoma the cause of the false passage, with the toothpick a mere incident thereto?

DISCUSSION

DR. F. H. COLBY: I enjoyed Dr. Leland's case very much indeed and congratulate him on his presentation. These cases of fistula formation between the bowel and bladder are sometimes extremely difficult to diagnose when the patient is first seen. It is usually a question as to whether the cause of the fistula is in the first place an inflammatory disease affecting usually the sigmoid, diverticulitis, or whether it is carcinoma. These two conditions account for practically all fistulae except the occasional congenital one, between bladder and bowel. While I was at the Brigham Hospital, there were two of these cases of fistulous formation between the bladder and the bowel. One patient complained of pneumaturia and the diagnosis was easy, of course. The other patient had a persistent cystitis much as in Dr. Leland's case. This patient was gone over very carefully, both on the general surgical and on the urological services, being on the urological service first. Cultures of the urine from kidneys and bladder and complete urological examination resulted in the diagnosis of chronic pyelitis for some time. Then the patient was given a barium enema and one of the nurses reported that barium had appeared in the urine. It is unusual to be able to find the fistula between the bowel and bladder diagnosed at cystoscopic examination unless the fistula is large. It is not frequently made in this manner following a barium enema, the barium appearing in the urine.

Carcinoma and diverticulitis apparently cause fistula formation in about the same number of incidences. Both of these patients had diverticulitis.

Divergence of the fecal stream by either a preliminary colostomy or cecostomy, and, following that, a repair of the damaged bowel either by resection or by a plastic operation, are usually the procedures that are followed and likely to be most successful. In one of these patients, the diseased mass could be removed by a plastic operation on the bowel and a Heineke-Mikulicz procedure done to restore the normal lumen of the bowel. In the other patient, following the colostomy and prolonged drainage of the bowel, resection was successfully performed, the fistula closed and the bowel eventually closed by a subsequent operation.

These cases are always interesting and should be always borne in mind by us as urologists, where a persistent cystitis without any evident cause is present.

DR. EDWARD J. O'BRIEN: There is a very close relation frequency of urination and these diverticula, especially when they become inflamed. I have seen several cases in the past four or five years where all the symptoms were those of the urinary bladder when the actual pathology was in the lower sigmoid or rectum. One individual about four years ago first came to me with some frequency of urination. I cystoscoped him but could not find a thing except four or five leucocytes per high power field in the urine. From time to time he complained of pain extending down the left cord into the scrotum. I had him x-rayed and the K. U. B. plates were absolutely negative. Bismuth enema was then done and a diagnosis of diverticulitis of the sigmoid was made. The man went along for about a year and a half without much trouble except for an occasional attack of pain and soreness along the cord extending into the left testicle. Two years ago last January he began to pass some gas through his urethra and that was followed by feces. I sent him to the hospital, cystoscoped him and found that he had an opening in the upper left side of the fundus of his bladder. On opening the abdomen there was a large inflamed mass that filled almost the entire pelvis. It was deemed inadvisable to do much exploration in this region so I did a cecostomy thus diverting the fecal stream. This was kept open for about three months with the large bowel irrigated daily with salt solution. The urine became clear and apparently the inflammation of the diverticula subsided. I then closed this cecostomy and the man has been very well since. The urine is clear and is negative except for an occasional leukocyte.

I also saw a case which was sent to me for obstruction of the prostate. There was no residual urine. I cystoscoped him and his bladder looked quite normal. A few weeks later he passed considerable blood by rectum. He was explored and found to have a massive carcinoma of the recto-sigmoid.

I mention these facts to show that where we see a lot of patients with frequency, and bladder findings are negative, we ought to be suspicious of some disease in the sigmoid or the pelvis.

DR. E. G. CRABTREE: I want to call attention to the possibility of demonstrating the sinus if you take an oblique cystogram. Most often these are connected with the rectum in the region of the sigmoid, either carcinomatous or inflammatory in origin, and in the right oblique view, when a dense solution is used, you nearly always see the sinus.

DR. CLYDE L. DEMING: There are no real papers on this subject and I am delighted that Dr. Leland presented this subject. The last paper on this subject was written by Hume about thirty years ago. With the advances of our diagnostic procedures, it seems to me it would be well for the urologists to bring this subject up to date, because certainly a great deal has happened in the last thirty years in regard to this subject.

DR. LELAND: These things usually run in threes. I had my second case last month. This patient had an admission diagnosis of indirect inguinal hernia, with a large mass low down in the abdomen. He, also, passed fecal material by urethra. He was *in extremis* at the time and no operative investigation could be made. At autopsy he showed a large diverticulum of the sigmoid which had, apparently, eroded through and had communicated with the bladder so that communication between the bladder and intestinal tract is more common than the textbooks would show.

A CASE OF CONGENITAL HYPERTROPHY OF THE VERUMONTANUM*

BY ROBERT R. BALDRIDGE, M.D.†

CONGENITAL hypertrophy of the verumontanum as a cause of urinary obstruction was first described by Bugbee and Wollstein in 1923[1]. The patient was a child three and one half years old. In 1924 the same authors[2] reported eight cases of posterior urethral obstruction from 4,903 autopsy records of the Babies' Hospital in New York City. Seven of these eight cases were demonstrated to have been due to hypertrophy of the verumontanum. They were all under one and one half years of age.

In 1927 Robinson[3] described the case of a male, seventeen years of age, who was brought to the hospital in uremic coma and who died shortly after admission. Autopsy showed posterior urethral obstruction due to an hypertrophied verumontanum.

Dodson and Lorraine[4], in 1931, reported another case in a 19 year old male, in whom the verumontanum produced almost complete obstruction of the posterior urethra. Appropriate treatment produced marked improvement. I have been unable to find reports of any other cases in the American or British Journals.

In 1934 Lowsley and Kirwin published a paper entitled "A Clinical and Pathological Study of Congenital Obstruction of the Urethra".[5] They confined their discussion entirely to valves of the posterior urethra, and did not mention hypertrophy of the verumontanum as a cause of obstruction. Their bibliography is extensive, and includes the work of Bugbee and Wollstein, and of Dodson and Lorraine.

Bugbee and Wollstein, and Robinson, studied the pathology of congenital hypertrophy of the verumontanum. In all cases examined the structure was identical with the normal, except for hypertrophy.

The symptoms and pathological findings in these cases are those of posterior urethral obstruction from any cause.

Symptoms developed early or late, depending on the degree of obstruction and on the degree of compensatory hypertrophy of the musculature above the point of obstruction.

Pathological findings, clinically or at autopsy, are essentially the same in the babies reported by Bugbee and Wollstein, and in the older patients of Robinson, and of Dodson and Lorraine. There is dilatation of the posterior urethra and bladder, and extensive bilateral hydro-ureter and hydronephrosis. The ureteral valves are incompetent.

Symptoms depend, naturally, on the stage of the disease, and on the presence or absence of infection. Those depending on the mechanics of the condition are difficulty in urination, dribbling, frequency, dysuria, but most important of all, intermittent lower abdominal distention and paradoxical incontinence.

The secondary group of symptoms, due to kidney failure, are those of uremia from any cause.

I wish to report the case of an eleven year old boy who was admitted to the Rhode Island Hospital on December 4, 1932. His chief complaints were hematuria and pain over the lower abdomen and also in both lumbar regions. Until two years before admission and, except for measles, chickenpox and whooping cough, his past history had been that of a healthy boy.

Two years before admission the patient first noticed that in the morning his lower abdomen appeared to be distended. This distention disappeared after voiding.

About one year before admission he ran and fell, belly down, on a sled. There was blood in the urine for twenty-four hours. He was seen by a physician, but no treatment was given. From the time of this accident until admission the patient developed progressive symptoms of difficulty in starting the urinary stream, perhaps followed in a few minutes by extreme urgency and incontinence. At times he was totally unable to void. He also noticed an intermittent enlargement of the lower abdomen, with pain in the bladder and in both flanks. The tumor, and likewise the pain, would disappear if he

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were able to void. On these occasions he passed an unusually large amount of urine. Paradoxical incontinence was a feature of the history, and bed wetting became a frequent occurrence. During this period the patient often stayed at home from school because of abdominal distention and pain. His appetite was poor.

Several hours before admission he had been wrestling with another boy, who threw him and fell on top of him. There was immediate severe pain in the lower abdomen, and in both kidney regions. Shortly afterward he passed bloody urine and was brought to the hospital.

Physical examination showed a well-developed and nourished boy, lying flat in bed, and in no severe distress. Pulse was 100 beats per minute, respirations 24, temperature 98.6 F. and blood pressure 110 systolic and 70 diastolic.

Examination was essentially normal except for the abdomen, which showed extreme tenderness and muscle spasm in the region of the bladder and of both kidneys. There was a mid-line abdominal tumor which extended from the symphysis pubis to above the umbilicus.

A soft rubber catheter (F. 16) was passed without difficulty, and the distended bladder gradually decompressed. The urine was grossly bloody for two weeks. There was a large trace of albumin, but no pus or casts. Culture was sterile.

Cystoscopy done on the second day after admission was not very satisfactory. The cystoscope (F. 18) met no resistance. The bladder wall lay in great folds which obscured vision, and when fully distended was so large that the cystoscopic lenses would not focus. We have had the same difficulty in vision when attempting to see objects on the other side of a large dark room by the light of a match.

In spite of the extreme degree of pathology demonstrated by cystoscopy and the intravenous urographs which were subsequently made, it is remarkable that on the fourth of December, two days after admission, examination of our patient's blood gave the following results:

Blood urea nitrogen— 17 mgm. per 100 cc. of blood.
" " creatinine—1.4 " " " " " "
Red blood count—5,000,000 cells per cu. mm.
Hemoglobin—80%
White blood count—9,240 cells per cu. mm.
Differential count:

Polymorphonuclear	65%
Lymphocytes	29%
Transitionals	6%

One week after admission an intravenous renal function test (phenolsulphonephthalein) showed the following:

Output of dye 1st half hour—	0%
" " " 2nd " " —	20%
" " " 3rd " " —	15%
" " " 4th " " —	5%
Total output in two hours	40%

Two weeks after admission, during which time the patient had been on permanent catheter drainage and his urine had become clear of blood, a second cystoscopy was done. The bladder wall was very thin and not trabeculated. It was generally congested. No diverticula, tumors or foreign bodies were seen. Both ureteral orifices were large and gaping. The bladder neck and prostatic urethra were dilated. The verumontanum was enormously enlarged, filling the entire undilated portion of the posterior urethra. It protruded into the bladder neck and could be pushed from side to side. There were no demonstrable valves. The tumor mass did not bleed easily. It was destroyed as completely as possible by electrocoagulation, and the patient continued on permanent catheter drainage.

During the next two weeks the catheter was removed several times, and much to our dismay the patient was unable to void. In the meantime he had developed a rise of temperature and a severe urethritis. Four weeks after admission, therefore, we performed a suprapubic cystotomy for more safe and adequate drainage. We found the bladder wall to be unusually thin, the bladder capacity very great and, to our satisfaction, no evidence of the tumor we had fulgurated, or any other urethral obstruction. We interpret the patient's inability to void as having been due to bladder atony and urethral infection.

After several weeks of suprapubic drainage, the sinus was allowed to heal and the patient was discharged, voiding normally, except for very slight incontinence, with a staphylococcus infection of his urine.

He has been seen repeatedly during the last two years, and many intravenous urographs have been made. As you see from the reproductions, there has been only a slow progress in the regaining of muscle tone of ureters and kidney pelves.

December—1932. Before treatment. Ureters difficult to visualize. Very large.

Nevertheless the last group of x-rays, taken two years after original treatment (retrograde pyelograms), demonstrate that although the kidneys and ureters have a capacity of fifteen cubic centimeters on each side, the emptying time is normal.

Renal function (intravenous phenolsulphonephthalein) done December 15, 1934, two years after treatment, and repeated for verification, gives the following:

Appearance time of dye (in bladder)—
3 minutes.

Output 1st half hour—	38%
" 2nd " " —	33%
" 3rd " " —	11%
" 4th " " —	4%
Total output in two hours	86%

Blood urea nitrogen (December 15, 1934)
13 mgm. per 100 cc. blood.

Urine cultures (December 15, 1934)
Right kidney—sterile
Left kidney—staphylococcus albus.

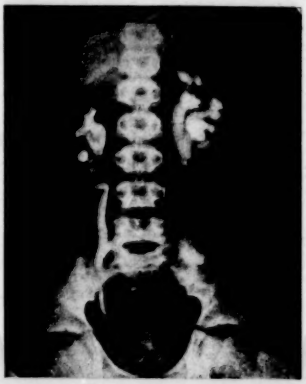
December—1932. Before treatment. Bladder at body of 4th lumbar vertebra.

June—1933. Note size of left ureter and large bladder capacity.

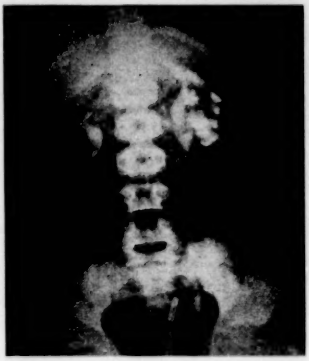
Urine examination (December 15, 1934)

Albumin—slight trace
Specific gravity—1016
Sugar—none
Microscopic—20 to 40 pus cells, with clumping, per high power field.

At the present time the patient is in excellent general health, and his infection is rapidly clearing up. There is no residual urine. Neither are there any symptoms referable to the genito-urinary system except a slight tendency toward incontinence in cold weather.



June—1934. 1½ years after treatment.



December—1934. 2 years after treatment.

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DISCUSSION

DR. F. H. COLBY: This is a very interesting case. Would the ketogenic diet be worth a trial?

DR. CLYDE L. DEMING: I should like to ask what the pathology of the verumontanum was?

DR. ERIC STONE: I was fortunate enough to see this picture. The tissue was destroyed by fulguration and it was impossible to get microscopical pic-

tures of the specimens. The appearance, however, of the tissue was in no way remarkable. It looked like a normal verumontanum except that it was out of all proportion to the usual structure. The mucous membranes were normal in appearance over it, and its general contour was what you would expect of the usual. The only abnormal feature was the tremendous enlargement of the tissue.

DR. BALDRIDGE: I believe that Dr. Colby's suggestion regarding the ketogenic diet is a good one, although staphylococcus infections do not respond to this form of treatment so frequently as do the bacillary types. I have nothing further to add to Dr. Stone's discussion.

So far as I have been able to find, this is the eleventh reported case of congenital hypertrophy of the verumontanum in the English literature.

HYDRONEPHROSIS—REPORT OF A CASE*

BY EARL S. MERRILL, M.D.†

URETERAL stricture is commonly bilateral and there is no very constant relation between the attacks of pain, the degree of hydronephrosis and the ability of the kidney to function as measured by phthalein excretion.

The following case report shows some interesting variations and illustrates the occasional necessity of ureteral dilatation before operation is attempted.

Present Illness: The patient, a man thirty-eight years of age, first consulted me September 30, 1933. He complained of repeated attacks of pain in the right kidney region. The pain radiated through to the front but not down along the ureter.

These attacks, some mild and some severe, had been recurring with gradually increasing frequency for about fifteen years. They were accompanied at times by nausea and vomiting. There had been no symptoms of urinary infection.

During the preceding year he had several times had a little pain in the left flank. This pain was intermittent, sharp but not severe and did not radiate.

Past History: Outside of the usual childhood diseases his health had been good.

Physical examination was essentially negative except that he was rather pale and thin.

Cystoscopic examination showed a normal bladder mucous membrane.

The ureteral orifices were normal and the ureters were catheterized easily. Sterile specimens of urine were normal on culture and routine examination.

The pyelogram of the right kidney showed very markedly dilated kidney pelvis and calyces.

On the left the injection was not very satisfactory but the kidney appeared to be essentially normal.

A phthalein test was done. The appearance time, following intravenous injection, was two minutes on the left. No phthalein appeared from the right. The left kidney secreted 15 per cent in one half hour.

Because of the low function test he was advised to have the left ureter dilated. A definite hang was found with a No. 10 bulb at about the junction of the upper and middle thirds of the ureter.

The ureter was dilated to a No. 10 Fr. four times before a No. 12 Fr. bulb could be passed. On November 6, 1933 about a month after dilatations were begun, the phthalein test was repeated. The left kidney secreted 20 per cent and the right kidney secreted 0.5 per cent in one half hour. Dilatations were continued and after six more treatments a No. 13 Fr. bulb was passed with some difficulty. On February 9, 1934 another phthalein test was done. The left kidney secreted 30 per cent in one half hour and the right kidney secreted just a trace. The patient at this time stated that he was feeling very much better although he had had several attacks of pain in the right kidney. He also reported that he had gained seven pounds in weight.

I continued dilating the ureter with a No. 13 bulb at about two week intervals.

April 17, 1934, six and one half months after beginning the dilatations, the phthalein test was repeated again. The left kidney secreted 35 per cent in one half hour and the right kidney just a trace.

May 19, 1934 the right kidney was removed. The patient stood the operation very well and convalescence was uneventful.

Since operation the left ureter has been dilated at intervals of about once every two months. The patient has gained about ten pounds in weight.

DISCUSSION

DR. E. G. CRABTREE: I have nothing in particular to say in discussion of Dr. Merrill's paper, except to emphasize the fact that one cannot estimate what reserve the kidney has until it is put to the test. This patient of Dr. Merrill's shows return to function that is perfectly surprising.

Perhaps this meeting would be a proper place to put on record a similar story, concerning a patient who has been under observation for a long period of time.

As resident at the Massachusetts General Hospital I operated on this woman for two large stones in a hydronephrotic kidney, which showed wide dilatation of the pelvis, thinning of the cortex and no function with phenolphthalein. Apparently the stones were the cause of the hydronephrosis. They were easily removed and the kidney was allowed to remain.

Then came the Great War at which time many physicians and surgeons were doing urology, often with little time spent on complete study. The surgeon who saw this patient with a cortical infection, due to staphylococcus, affecting her remaining kidney, asked her if the opposite kidney had been removed at my operation. When she replied that it had not been removed, he proceeded to do a nephrectomy on the previously normal kidney. After operation there were two or three years of very stormy renal difficulties. Eventually she returned to good health, married and has had three pregnancies, typhoid fever and pneumonia and was still living and seen at the Boston Lying-In hospital last year.

DR. CLYDE L. DEMING: Dr. Merrill's case is an extremely interesting one. I am going to report briefly a case of a young woman with one child who came to us with extreme pain on one side due to a large hydronephrosis with very little function. On the other side, we found an uninfected hypoplastic kidney with only a trace of phthalein function, so

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that we found ourselves practically in the same position as did Dr. Merrill. One kidney seemed to do most of the work and the opposite one was practically of no value. Necessity demanded that we save that kidney in some way. We were obliged to do a plastic on the hydronephrosis and she recovered very well. Others of you have probably had somewhat similar experiences, finding yourselves in the position that we have, when necessity demands that we save the kidney one way or another. I am sure we are forced to use conservative means under these conditions. Dr. Merrill is to be congratulated on the conservative way in which he handled his case.

TRANSURETHRAL RESECTION OF THE INTERNAL SPHINCTER IN A CERTAIN TYPE OF CORD BLADDER*

BY EDWARD L. PEIRSON, JR., M.D.†

THE classical cord bladder shows a marked loss of tone and sensation of the bladder wall with relaxation and funnelling of the bladder neck. There is, however, another type of neuropathic bladder which is seen in the presence of a minimal cord lesion. In this type, the tone and sensation of the bladder wall are maintained and the sphincters are spastic instead of relaxed. In such a case, one finds retention of urine due, not so much to failure of the bladder musculature to contract and expel the urine, as to failure of the sphincters to open properly during the act of voiding. The continued effort of the bladder to force out the urine through an uncoöperative sphincter produces in the end, exactly the same state of affairs as would be produced by a bladder trying to force out the urine through an obstructing prostate or a contracted bladder neck.

My experience with this type of cord lesion has been limited to six proved cases, in two of which I had an opportunity to examine the bladder at autopsy. An operation was performed on each of the six cases; the three advanced cases being treated by suprapubic cystotomy and three favorable ones by transurethral resection of the internal sphincter. The actual neurological diagnosis fortunately is not of great importance as we are interested in the functioning of the bladder and not in the cause of this dysfunction. It appears, however, that if the cord damage is extensive, a different type of bladder lesion with loss of bladder tone will develop. The neurological diagnosis was central nervous system syphilis four cases, spina bifida one case, and poliomyelitis one case.

The outstanding symptom, which brought all these cases to the hospital, was either acute or chronic retention of urine. The outstanding finding upon examination was a bladder which appeared characteristic of obstruction at the bladder neck without any visual evidence of organic obstruction. If the condition had been present for a long time, as it had in the majority of the cases, a coarsely trabeculated bladder was found. In the three cases in which I had a chance to examine the bladder wall, I found it very much thickened and identical with the type of bladder seen in long-standing prostatic obstruction. This is very different from the thin atonic bladder wall classically described as associated with cord lesions. The cystoscopic picture differed from organic obstruction in certain ways. Usually the interureteric ridge did not seem to share in the general hypertrophy. There was at least slight dilatation of the posterior urethra and as one looked inwards from the region of the verumontanum, the internal vesical orifice appeared at least as wide open as normal. Five of the six cases showed definite dilatation of the upper urinary tract from back pressure and one case had a moderate-sized diverticulum.

There is obviously great danger in confusing this condition with actual organic obstruction such as median bar or contraction of the bladder neck. In fact, I am inclined to believe that many cases which have been considered contracted bladder necks were in reality spastic internal sphincters. In certain cases, at least, it is impossible to make the differential diagnosis unless the internal sphincter can be directly examined with the finger, either under general anesthesia or at autopsy, as upon opening the bladder under local anesthesia the spastic internal sphincter may feel identical with a contracted bladder neck due to a chronic inflammatory condition. Likewise, as the nerve injury to the spinal cord is usually not extensive, the condition may not be obvious. Furthermore, there is some evidence to show that it may be possible to have retention of urine due to spasm of the internal sphincter without any cord lesion at all. Certainly, a very similar, although a transitory condition is frequently seen following a simple abdominal operation. In other cases, however, I have found it possible to demonstrate, without opening the bladder, that the retention of urine was due to sphincter spasm (or perhaps more accurately, to failure of the internal sphincter to open properly), and not to organic obstruction.

In spite of the fact that some bladder disturbance is found in the majority of cases suffering from disease of the spinal cord, the investigation of the urinary tract in these cases has frequently been overlooked. Often, the physician in charge of the case has failed to realize that the bladder symptoms may be the first and only sign of spinal cord disease, and that the management of the retention of urine may be the

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most important factor in the care of these patients. The seriousness of this condition is well shown in our series. Four of our cases were extremely sick from renal insufficiency and sepsis of the upper urinary tract. The two cases that had an N. P. N. of more than 100 before operation both eventually died in spite of some temporary improvement following suprapubic cystotomy. The history of these six cases show very clearly that treatment by occasional catheterization leads invariably to disaster. The retention of urine must, in these cases, as in cases of prostatic obstruction, be treated in some way which will give adequate relief of the back pressure on the upper urinary tract.

If the case has not been neglected, I believe the retention of urine can be cared for by resection of the internal sphincter. I should like to report briefly the last case which I have operated on by this method as this case gives a very fair picture of the condition and is very similar to the other cases.

This case was a man, forty-two years of age, who entered the Lynn Hospital for the last time on October 26, 1934. At the age of three, the patient had what apparently was anterior polio which had left

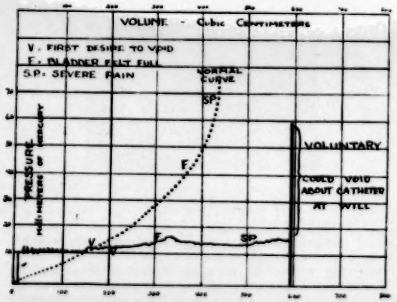

PLATE 1. Cystometerogram of the case described in this paper (August, 1932). It shows some lack of tone of the bladder musculature.

him a cripple with complete motor paralysis of both legs except for the big toe on the right foot. As a result, there was little or no development of the legs, marked scoliosis, and deformity of the pelvis. Sensation of the legs, sexual and urinary functions had been normal although the urinary stream had been poor. The patient's first urinary symptoms developed in March, 1932. At this time, he suddenly developed acute retention. I carried out a very careful urological examination and was unable to demonstrate any pathology in the urinary tract. After a few catheterizations, he was again able to void. During the following two and one-half years, the patient developed more and more urinary symptoms, the outstanding one being repeated attacks of acute retention which always subsided and allowed the patient to empty his bladder after a period of catheter drainage. During this period, there was increasing frequency and difficulty in urination. A diagnosis of cord bladder was made on purely negative evidence. During this period of time he was repeatedly admitted to the Lynn Hospital and was also studied by Dr. Richard Chute at the Baker Memorial Hospital where a cystometerogram was done which showed normal bladder sensation with some loss of tone of the bladder musculature. (Plate 1.)

On the last admission to the hospital in October, 1934, he had acute retention of forty-eight hours duration with a greatly distended bladder. The prostate was small and indistinct and the tone of the rectal sphincter was normal. He was put on catheter drainage for two days, at the end of which time, I cystoscoped him. It was surprising to find at this time that his bladder which, two days before, had held over forty ounces, would now hold less than two and that he was able to empty his bladder completely. This marked variation, both of the bladder tone and capacity, and also in the patient's ability to void, is, I believe, characteristic of the functional condition under consideration. Cystoscopic examination showed some trabeculation

PLATE 2. Urethrogram of a normal individual taken by injecting opaque solution backwards from the meatus.

of the bladder with considerable inflammation. No prostatic enlargement was seen and the posterior urethra was not elongated. The posterior urethra, which previously had appeared normal, now appeared definitely dilated but the internal sphincter showed only slight dilation. Upon looking back toward the bladder neck with a panendoscope from the region of the verumontanum, one could see clearly through the internal vesical orifice which was open wider than normally. The dilation of the posterior urethra made the floor of the urethra lower than the bladder neck and consequently the internal sphincter stood out prominently and one could easily have mistaken the sphincter for a median bar. Intravenous pyelograms made at this time and compared with plates taken two years before showed that marked dilation of the upper urinary tract had occurred in the meantime. There had, likewise, been a reduction in the urinary function.

At this time, I obtained some urethrograms which I now wish to show. I previously found that urethrograms taken by injecting opaque solution backwards through the urethra often gave one a very false idea of the condition in the posterior urethra and bladder neck. Accordingly, acting upon a suggestion of Dr. E. G. Crabtree, these urethrograms were obtained by filling the patient's bladder with an opaque

solution and taking the picture while the patient was voiding. For this purpose, I devised an emulsion of mineral oil acacia and sodium iodide which was very satisfactory and much more economical than lipiodol. Plate 2 is a normal urethrogram taken by injecting the solution backwards from the meatus in the usual way. I wish you would compare it with plate 3, which is a urethrogram of the same normal individual taken during the act of voiding.

PLATE 3. Urethrogram of the same normal individual (Plate 2) taken while the patient was actually voiding. If one compares this with the previous plate, one observes the changes which normally occur at the bladder neck during the act of micturition.

You will notice that in plate 3 the bladder neck has opened widely to form a funnel, and that this has apparently been accomplished by a pulling downwards of the bladder neck so that the area of constriction in the internal sphincter and also the bladder floor is considerably lower than before. Presumably, this has been accomplished by

PLATE 4. Urethrogram of the case described in this paper taken during the act of voiding. One observes that the spastic internal sphincter obstructs the bladder neck. See text.

the action of the muscles which run from the trigone down the urethra. This demonstrates the importance of the fact, that the opening of the internal sphincter is an active and not a passive phenomenon and explains the retention of urine in these cases. It also explains why the retention is relieved by resection of the sphincter even if the sphincter is somewhat dilated before operation. In this plate both sphincters are well relaxed and there is a large stream passing through the urethra.

The third urethrogram (plate 4) is of the patient under discussion taken during the act of voiding. In spite of this, one observes that the internal sphincter has not relaxed. The bladder neck has not been pulled down and funnelled out. The posterior urethra appears dilated and the external sphincter, in spite of the fact that the patient is voiding, is not relaxed.

I think one can see from this how easily one might confuse the spastic, internal sphincter with a median bar on cystoscopic examination. In fact, the failure of the internal sphincter to open actively has produced a condition nearly identical with actual obstruction at the bladder neck. The marked dilation of the posterior urethra and the lack of opening and depression of the bladder neck, seem to me, to be very striking and to prove conclusively that the retention of urine is due to spasm of the sphincter muscles and not to an obstructive lesion. Unfortunately, this plate is not very good, as the marked deformity of the pelvis made it difficult to get the patient squarely centered on the table.

The last plate (plate 5) is a urethrogram on this

PLATE 5. Urethrogram of the same patient obtained six days after operation. It shows the artificial funnelling of the bladder neck which has been produced by resection of the internal sphincter.

same patient taken six days after transurethral resection of the internal sphincter. One observes here that action of the internal sphincter has been destroyed and the posterior urethra has become continuous with the bladder. This artificial funnelling of the bladder neck has produced a condition not very different from a normal state of affairs during micturition as shown in the previous plate, the main difference being that the bladder base has not been pulled down. The external sphincter is still somewhat spastic but the greater force of the urinary stream has caused it to open more widely than before operation.

Following the taking of these urethrograms, the patient was taken off catheter drainage and observed for a week. For two days he was able to void well. Then he began to have marked frequency and difficulty in voiding. The stream became smaller and he passed only a small amount at a time. The residual urine increased daily until at the end of the week it amounted to six ounces. On November 6, I resected his internal sphincter with a McCarthy resectoscope removing three large pieces from the posterior and posterior lateral aspects. The pathological examination showed numerous muscle bundles so arranged as to suggest sphincter muscles. Immediately beneath the mucosa there were large clumps of lymphocytes and plasma cells.

Twenty-four hours after resection, the patient was able to void easily and passed a large and more forceful stream than he had ever done in his life. He has continued, to date, to void easily and freely without residual urine. He has no incontinence. He gets up once at night and passes his urine about four times a day. A check-up cystoscopic examination shows the same funnelling of the bladder neck as is seen in the postoperative urethrogram. The posterior urethra and bladder appear continuous.

The two other cases in which I have carried out this operation have been quite similar to the one reported. In both cases, the neurological diagnosis was tabes-paresis. Both cases came to the hospital with a large residual urine and were preoperatively treated by catheter drainage with, as is common in these cases, marked improvement in the bladder decompensation. It was not sufficient, however, to

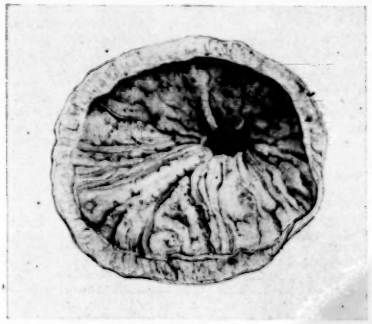

PLATE 6. Drawing of the bladder neck from an autopsy specimen of a patient with central nervous system syphilis.
The history and cystoscopic findings were similar to the case reported in this paper. When the bladder neck was examined at the time of suprapubic cystotomy under local anesthesia, it was found to be tightly closed and felt to the examining finger like a contracted bladder neck. This autopsy specimen shows, however, that the contraction of the bladder neck was neurogenic and not organic in origin. At autopsy, the bladder wall was greatly thickened, there was marked dilation of the upper urinary tract and a diverticulum was present.

allow them to empty their bladders even temporarily. The first case was operated on seven months ago. He came to the Salem Hospital with acute retention and a greatly distended bladder. He had had frequency and difficulty of urination for three years. The urinary stream was small and he was known to have had some residual urine for several years. The findings were very similar to the case just reported. Following resection of the internal sphincter, he has been able to void normally and has had no residual. He has slight frequency and urgency but no incontinence. The third case was operated on four months ago. He came to the Salem Hospital with twenty-four ounces of extremely foul residual urine and a bilateral pyonephrosis which was caused by the back pressure. There was more loss of bladder tone and more dilatation of the posterior urethra and internal sphincter in this case than in the other two. He was uremic and was running a septic temperature. After a period of catheter drainage a transurethral resection of the internal sphincter was carried out. His urinary stream has never been so forceful as in the other cases because of loss of bladder tone but he has been able to empty his bladder. In spite of the fact that he did not carry out the antiluetic treatment after leaving the hospital, he gained twenty pounds in two months and was able to return to work, which I think shows the great improvement which can be obtained with adequate bladder drainage in these cases.

As regards the operation, it is extremely simple and very much more efficient in this type of case than any operation on the sympathetic nervous system. Such a small amount of tissue needs to be removed that it is truly a minor procedure. If the tone of the external sphincter is maintained (which can easily be determined by urethrograms or by observation) there is no danger of incontinenee.

There is, however, the theoretical possibility that there may be sufficient spasm of the external sphincter to prevent voiding even after resection of the internal sphincter. It is my experience, however, that if the internal sphincter is not markedly relaxed and the bladder tone has been sufficiently maintained, the operation will be successful. If, however, study of the case shows marked loss of tone of the musculature of the bladder wall or marked relaxation of either sphincter, this operation is contraindicated. As regards the permanency of the result, I know nothing, as these cases have been followed only a short time. It seems reasonable to suppose that if the progress of the disease in the spinal cord cannot be checked, they may develop further urinary complications. On the other hand, if no further damage to the nerves of the bladder occurs, I believe the results will be permanent, and even if this is not the case, the improvement in these patients to date has been sufficient to justify the procedure. This type of patient must have adequate bladder drainage if his health is to be preserved. This can be obtained either by resection of the internal sphincter or by permanent suprapubic cystotomy. It seems logical to save the latter procedure for those cases which cannot be handled by resection of the internal sphincter.

DISCUSSION

DR. RICHARD CHUTE: I would like to compliment Dr. Peirson on his very real and ingenious contribution to this subject. I have been interested in this for some years and I do not think there is anything more puzzling than one of these neurological bladders.

He spoke of one thing that I think is a conception which a lot of people have not yet acquired, that is this business of decompensation and recompensation of the bladder. You take a bladder that has decompensated due to some sort of obstruction, either neurogenic or prostatic and as in Dr. Peirson's case which had forty ounces of residual, you rest it with catheter drainage and allow it to recompensate, and on recompensation it will only hold two ounces. If we bear that conception in mind, it will often straighten out some of our bladder difficulties. Dr. Peirson's third case, where the patient, apparently, did not have much pain in his bladder, I should

think, if he started running a residual it might be a good thing to do a sympathectomy in combination with this, but, as Dr. Peirson shows this is so much simpler and easier, I should think it would be a good thing to try.

I was wondering about the rectal sphincter. Was that also spastic?

DR. F. H. COLBY: This communication of Dr. Peirson's is excellent. It sounds a little too good to be true, that is all. These cases have always been difficult problems.

I wish to ask Dr. Peirson if during the course of his endoscopic examination, viewing the internal sphincter, he asked these patients to try to void voluntarily and then stop voiding, whether he saw any movement of the internal sphincter, whether they could relax it or whether they could contract it to any extent.

DR. C. J. E. KICKHAM: Relative to the paper of Dr. Peirson and the question of infection in his group of cases, I would like to ask whether the intractable chronic infection so frequently encountered in the urinary tract of the cord bladder individual affected the postoperative course in his cases. Sepsis is the most common complication of transurethral resection and also the most important contraindication. In view of this, it seems to me that this procedure might be quite hazardous in this type of case.

DR. G. G. SMITH: I have been tremendously interested in this paper of Dr. Peirson's. There is one point I wish he would comment on in discussion. In the cases of tabes that I have cystoscoped, I have always seen the bladder neck wide open so that even with an observation cystoscope you could look around the posterior urethra. In other words, this funnel-shaped bladder neck seems to have already developed. I would like to have him put me right about this and tell me whether they were more advanced cases or why I found that appearance to exist.

DR. CLYDE L. DEMING: There is one point which I do not quite comprehend and that is, why can one look through the internal sphincter of the bladder from the posterior urethra in these cases? I do not doubt that this is true, but if it is true why should not the patient void? His urograms are convincing that there is something wrong.

DR. E. L. PEIRSON: As regards Dr. Colby's statement, I should say that I tried seeing whether they could relax the bladder neck when I looked in with the endoscope. I also tried it in one or two who were normal, and I was not convinced that I could tell one way or the other. I think, as Dr. Colby pointed out, these results are much too good to last and I think I brought out the point that I do not know whether they will last. We do know, however, with this type of cord bladder with increased bladder pressure that the patient will go on and die from sepsis and back pressure on the upper urinary tract if that pressure is not adequately relieved. Something has to be done for them. It seems to me it is entirely justifiable to do this operation for if it fails they can still have the suprapubic cystotomy, but I think that treatment by pity and occasional catheterization is not justifiable.

As regards infection as a complication, I think it is very important. Because of the severe infection I did a suprapubic cystotomy in three of these cases. In the one case which I resected which had a pretty bad infection, the infection had been fairly well cleared by catheter drainage before the operation was undertaken. Such a small amount of tissue needs to be removed in this operation that infection does not play such a serious part as it does in resecting an adenoma of the prostate.

As regards Dr. Smith's question about the dilated bladder neck, I think when you consider the extreme complexity, the neurophysiology of the bladder and in addition, consider the diffuse and scattered nature of the lesions of the spinal cord that it is quite easy to conceive that by placing your lesions in the spinal cord at the proper place you can produce almost any type of lesion of the bladder. You can have relaxation of the sphincter or spasm, you can have increased or reduced tone of the bladder wall. At the present time, I have in the Salem hospital a case exactly like the one Dr. Smith speaks of, with acute retention, a large residual urine, infection and marked funnelling of the bladder neck, with spasm of the external sphincter so that he is unable to void. That shows up nicely in the urethrograms when we fill up the bladder and he tries to hold it, the urine comes down to the external sphincter. In other words, the whole posterior urethra is tremendously dilated. That type of case is obviously unsuited for this form of treatment.

Perhaps the confusion is that I was speaking of looking back from the verumontanum, using the McCarthy panendoscope with the fore-oblique vision. You look back from the verumontanum into the bladder neck just as you can with the resectoscope. Of course, the prostatic urethra in these cases is very short and you can see the bladder quite clearly with the panendoscope. The reason they can void after you have resected the internal sphincter, although the internal sphincter was open before you resected it, is, I think, quite clearly shown in the third urethrogram which I showed. In this urethrogram the internal sphincter stood out quite clearly as a definite band at the bladder neck which would cause obstruction to the outflow of urine just as a chronically inflamed, hard, and rigid bladder neck causes obstruction. In either case, the opening of the bladder neck may be of sufficient calibre to allow the outflow of urine, yet retention may exist because of the bar of tissue posterior to the internal meatus which fails to relax at the time of micturition.

The surprising thing was, it seems to me, that the other neurological findings were very slight. In some cases the rectal sphincter was relaxed. In some we found a few other neurological signs such as absent or hyperactive knee jerks, absent corneal reflexes in two cases, and fixed pupils in two cases. But by and large the neurological signs were quite striking by their absence, although the findings in the spinal fluid were very definite in all the syphilitic cases.

THE RECURRENCE OF BENIGN OBSTRUCTING PROSTATES YEARS AFTER PROSTATECTOMY*

BY RICHARD CHUTE, M.D.†

A SUCCESSFUL prostatectomy on a benign hypertrophied obstructing prostate has always carried with it the thought of a permanent cure, and urological surgeons are wont to say to their patients, "Well, old man, it's all out now, and it'll never bother you any more." They are right in more than 99 per cent of the cases, but not in 100 per cent, for on rare occasions regrowth of the benign hyperplastic prostatic tissue will take place to the extent of causing typical prostatic obstruction again. I would like to report two such patients from whom I have removed good-sized, benign, obstructing, "regrown" prostates in the last few months, patients on whom my father had performed successful prostatectomies ten years and eighteen years before respectively.

The first patient was fifty-nine years old when my father operated on him in 1924. At that time he gave a five months' history of difficult urination with frequent desire, etc., and he had six ounces of infected residual. By rectum his prostate felt perfectly benign, and was not markedly enlarged. A one-stage suprapubic prostatectomy was done, and three small masses, largely intraurethral, were enucleated in the usual manner without any difficulty. The microscopic report was benign hypertrophy. The patient made a satisfactory recovery, and had been without urinary symptoms for ten years until a few months before I was called to see him last August after two episodes of acute retention. His prostate by rectum felt somewhat larger than normal and absolutely benign. Cystoscopy showed a marked anterior cleft and more or less encroachment of the prostate in the median region, also marked trabeculation. Eye reflexes and knee jerks were normal. After the usual preparation, I did a one-stage suprapubic prostatectomy, easily enucleating a moderate-sized benign prostate in one piece consisting of both lateral lobes. The pathological report on this was benign hypertrophy. The patient made a satisfactory convalescence, and I have seen him recently. He voids with perfect control, and has practically no residual urine.

The second patient was only forty-seven years old when my father operated on him in 1916. Father saw him in his second episode of acute retention with his bladder up to his umbilicus and withdrew thirty-one ounces of turbid urine. The patient gave a history of painful, difficult urination for about a year; his stream was very slow and painful to start, and he passed only a little urine at one time. His prostate by rectum felt slightly enlarged and benign. Cystoscopy showed the prostate jutting in on all sides, especially on the right, with some trabeculation, and also a small diverticulum of the bladder. Knee jerks and eye reflexes were normal. Father did a one-stage prostatectomy, shelling out a somewhat enlarged prostate in the usual manner, and not touching the small diverticulum. The patient made a satisfactory convalescence, and ended by voiding with perfect control, with only one-half ounce of residual urine. After the operation, the patient was fine for nearly fifteen years, but in the last three years had had mild symptoms of recurrent prostatism, a few ounces of residual urine had begun to accumulate, and rectal examination revealed considerable benign regrowth of his prostate. Last October I was called to see him in acute retention, and withdrew twenty-six ounces of residual. His prostate by rectum was considerably enlarged, and it was necessary to depress the cystoscope markedly in order to pass it over the raised bladder outlet, and cystoscopy showed considerable prostatic tissue jutting in the median and lateral regions. Knee jerks and eye reflexes were normal. I did a two-stage suprapubic prostatectomy on him, easily enucleating in one piece a good sized prostate which weighed fifty grams. The pathological report was benign hypertrophy. He made a satisfactory recovery, and I have seen him within a month. He voids with perfect control, and empties his bladder practically completely.

These two cases meet the requirements for genuine benign recurrence so well expressed by Victor Blum of Vienna[1], who feels that only those cases should be classed as genuine recurrences in which, after a properly and carefully performed perineal or suprapubic prostatectomy, and after an interval of several years, during which there is normal urination, the symptoms of prostatic obstruction reappear, and a recurrent prostatic hypertrophy is definitely present by rectal examination and cystoscopic inspection *or* through the opened bladder, and in which there is not the slightest suspicion of malignancy.

I have looked through a good deal of the world literature on this subject, and have found less than fifty cases which fulfill the above requirements for genuine recurrence of benign obstruction. A good many cases have been reported in which considerable regrowth of the prostate some time after prostatectomy has been evident on rectal palpation or on postmortem examination, but there have been very few of these cases reported which gave urinary obstruction again during life. We have not the time to consider the various theories as to the place of origin, and mechanism of growth of benign prostatic hypertrophy but, according to the best opinions at the present time, the hyperplastic growth probably starts in the submucous periurethral groups of prostatic glands situated in the region of the neck of the bladder. As these centrally placed glands of the prostate undergo hypertrophy and grow, they form an expanding adenomatous mass which presses on the outer layer of prostatic glands, which gradually atrophy and become flattened, and form the so-

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called "surgical capsule" of the prostate. The ordinary enucleation of the prostate is not really a complete prostatectomy; it is merely the separation of one part of the prostate from another part of the prostate along an easy plane of cleavage. It is the shelling-out of a tumor mass composed of the hyperplastic central glands of the prostate from the so-called "capsule," which is composed of the flattened and atrophic outer prostatic glands and is left behind. This is a well-known fact of long standing. As to the theories as to the cause of regrowth: one is that the flattened atrophic outer glands of the prostate which formed the capsule may, in the course of time, take on new life, proliferate, and occasionally undergo enough hyperplasia to form another obstructing growth, after the removal of the adenoma of the central glands which had been making pressure on them. It has also been shown by various men[2, 3, 4] that after prostatectomy, very small nodules of hyperplastic tissue, often only recognizable under the microscope, can frequently be found in the surgical capsule, and it is thought by some that these hyperplastic nodules continue their growth and eventually may produce a recurrent obstructing mass.

The average age of the patients in my series was sixty years at the time of their first operation. This is more than five years younger than the age of the average prostatic operated upon in the Massachusetts General[5] and Johns Hopkins[6] hospitals, and it may be that it signifies that these patients have prostatic cells which have greater vitality and tendency to proliferate than the average, so that they get their masters into trouble five years earlier than the average prostatic, and then are hard to eradicate, and stubbornly regrow again after operation like hardy perennials. It seems likely that there may be some endocrine gland factor, not understood at present. The clinical recurrences in my series appeared on an average of seven and a half years after the first operation.

SUMMARY

The paucity of reports of true recurrent benign prostatic obstruction is very hard to explain, considering the thousands of prostatectomies that have been performed since the turn of the century, but I do wish to call your attention to the fact that genuine benign recurrences following prostatectomy occasionally occur, and to report two personal cases that recurred ten years and eighteen years respectively after their original operations.

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DISCUSSION

DR. E. L. MERRITT: I recall that several years ago at a clinic given by Dr. Squier in New York, he called attention to the necessity of removing all of the small spheroidal bodies, as he called them, at the time one did a prostatectomy and in the particular case he was operating upon at that time he removed a fairly large, adenomatous prostate and then he reached down into the prostatic fossa and removed quite a long chain of these hyperplastic nodules, as Dr. Chute called them. Several times since then, in doing prostatectomies, I have reached down into that fossa following the removal of the gland, finding quite a long chain of these small nodules and I have recalled that Dr. Squier brought out the fact that if they were left he felt quite confident that they would cause recurrence. I wonder how the other men feel about that.

DR. HAROLD S. BACKUS: I was especially interested in this paper because when I first took up the study of Urology there was a very prominent surgeon who did a secondary prostatectomy ten years after the first prostatectomy, for which he got $5000. That interested me, thinking I might be able to make a living doing Urology. Also, about fifteen years ago before Prof. Hugh Cabot went west urologically, one of the first things I heard him say, in one of his lectures, was, "We do not remove the prostate, we just remove the adenomatous growths and there is no reason why they cannot grow again." For that reason I was interested to know that they do grow again.

I find, in doing prostatectomies, that there is quite often considerable prostatic tissue left after the growths have been enucleated. I do not try to get it out, if it causes no obstruction. However, I believe this tissue may hypertrophy and cause trouble in a few cases.

DR. CLYDE L. DEMING: In New Haven we have been studying prostatic hypertrophy for a couple of years and we have a hunch as to why these recur. We feel that prostatic hypertrophy arises from the glands of the posterior urethra, that it arises from the lateral lobes of the true prostatic gland and that it arises from the glands between the verumontanum and neck of the bladder deep in the prostate. It is reasonable to expect that a greater number of these hypertrophies arise from the glands of the posterior urethra. If these are removed primarily we do not in any way interfere with the other multiple foci for prostatic development deep in the prostatic gland.

DR. CHUTE: As to Dr. Merritt's remarks, I think it is a very good point about the spheroidal bodies which you occasionally see and I, for one, from now on am going to try to do more one-stage suprapubic prostatectomies because when you reach down through the typical sinus in the second stage operation while you can do a pretty good job, you don't have the flexibility that you do with a one-stage prostatectomy when you have things wide open and can view and explore the prostatic bed pretty thoroughly, and I think on the basis of this, I shall try to do more one-stage operations.

Dr. Backus speaks of the matter of the big fee. Of course, there is the possibility that a very mean patient who had a recurrence after a few years

might sue you, especially if he had paid a good fee in the first place. He might claim that the operation had been badly done, and it is conceivable it might cost you quite a bit and also a loss of reputation if it got into the newspapers.

As for Dr. Deming's remarks, that discussion is so involved that I won't try to go into it. I was merely stating things as I saw them, but I know that Thompson Walker in London and others would agree with you on that. But the big wonder of it all to me is the fact that there must have been 100,000 prostatectomies done since 1900 and I can only find fifty reports of genuine recurrences in the literature.

CYSTS OF THE TESTICLE*

BY RALPH H. JENKINS, M.D.,† AND CLYDE L. DEMING, M.D.†

CYSTS of the testicle are classified into cysts of the tunica albuginea and cysts of the testicle. They are exceedingly rare. In reviewing the literature it is difficult to separate simple cysts of the testicle from cystic neoplasms. Sir Astley Cooper[3] in 1845, described the gross anatomy of teratoma of the testicle under the term of hydatid disease. This article was elaborately illustrated and four cases of simple cysts were described. Curling[4] in 1853, divided the growths into cystic, relatively benign, and solid malignant tumors. He described two such cases as simple cysts. Ewing[5] in studying the reports of Cooper and Curling felt that their cysts were of neoplastic origin. Succeeding accounts were less accurate in differentiating cyst of the testicle and cystic neoplasms. Cases were described, however, by Hochenegg[7], 1885 and Walliman[10], 1911. Albert[1] 1893, mentions three cysts of the testicle in 6000 autopsies.

C. K. Smith[9], in 1919, reported a large cyst of the testicle which he believed followed gonorrhea. He suggested that the etiology was an occlusion of the vas deferens and epididymis following epididymitis resulting in a cystic condition of the epididymis and secondarily involving the testicle.

Barach[2], in 1919, reports a very interesting case of cyst of the testicle in a dog. He considered trauma as the etiological factor as this dog was run over by an automobile four or five years previous to his operation.

K. Frater[6], in 1929, reported and illustrated a case of cyst of the tunica albuginea. The cyst measuring 1 cm. in diameter, contained clear fluid and was located in the anterior surface of the left testicle near the upper pole. The walls of the cavity appeared to be complete. The rest of the testicle showed little change. The epididymis was normal. The inner surface of the wall was lined in some parts by a single layer of cells and in others two or more. The nuclei of these cells were slightly ovoid. There was no history of gonorrhea, injury or abnormality of the testicle. The urine contained four pus cells to the high power field. The Wassermann reaction on the blood was negative. The patient died from pernicious anemia.

Simple cysts of the testicle should not be confused with an encysted hydrocele of the body of the testicle. The latter is a small collection of serous fluid beneath the visceral portion of the tunica vaginalis, which, according to Rose and Carless[8], is probably due to the dilatation of the lymphatic spaces and has no clinical significance.

The case reported here is a cyst of the testicle.

A man twenty-eight years of age was referred by his family physician to Dr. Clyde Deming for impotency. He had indulged in an active sexual life which consisted of intercourse five times daily since his marriage four years ago. During the past two months he was only able to have intercourse about once in five days due to the lack of erections. There were no premature ejaculations and no other symptoms other than that he complained of being weak and tired following intercourse. There was no history of gonorrhea, injury or abnormality of the testicles.

Our examination was entirely negative except that a hard, painless, somewhat irregular mass measuring 2 x 3 cm. was found involving the lower lobe of the right testicle. This mass did not transmit light. The right epididymis and vas deferens were normal in size and consistency. The left scrotal contents were normal. The urine was normal. The prostatic and vesicular fluids were normal. The Wassermann reaction on the blood was negative.

The patient was seen a few weeks later and the mass in the testicle had apparently increased in size. Our diagnosis was tumor of the testicle. An orchidectomy was performed under local anesthesia and the patient made an uneventful recovery.

Our surgical pathologist, Dr. Béla Halpert, gave the following pathological report:

"*Gross:* The specimen consists of the right testicle, epididymis and a part of the spermatic cord enclosed in the tunica vaginalis communis. The cavity of the tunica vaginalis contains no excessive fluid and its surfaces are smooth and shiny. The testicle and epididymis are of normal size and shape and measure 5.5 x 3 x 2 cm. In the lower pole of the testicle there is a firm area beneath the capsule. The cut surfaces present the usual pattern, except in the lower third where an amorphous laminated structure occupies an oval area 24 x 16 mm. The area is well circumscribed, appears to have a capsule, and is soft in the center, and quite firm in the periphery. It is grey and translucent while the rest of the surface is light orange-yellow.

"*Microscopic:* The seminiferous tubules are of

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Read at the meeting of the New England Branch of the American Urological Association, Boston, February 14, 1935.

†Jenkins, Ralph H.—Assistant Clinical Professor of Urology, Yale University School of Medicine. Deming, Clyde L.—Clinical Professor of Urology, Yale University School of Medicine. For records and addresses of authors see "This Week's Issue," page 75.

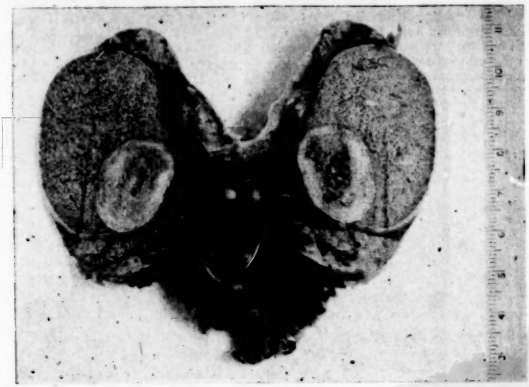

FIG. 1. Cross section of cyst in testicle (natural size).

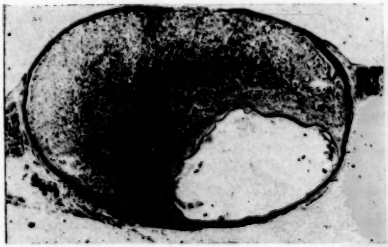

FIG. 2. Cyst completely invested by a fibrous connective tissue capsule in the testicle (slightly enlarged).

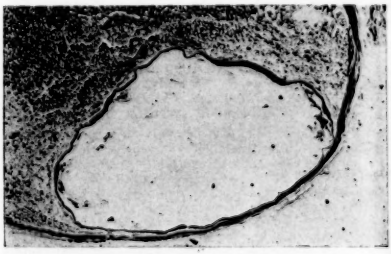

FIG. 3. Cyst wall (3+ ×).

good size and fairly close to one another with little intervening stroma and groups of interstitial cells. Spermia are scant. Beneath the tunica albuginea, which is of the usual structure, there is an oval area surrounded by a fibrous connective tissue capsule. An occasional tubule near the capsule appears flattened. This capsule is of the same structure as the tunica albuginea but only about one-third as thick. Deep blue staining granules are seen along the margin on the inner surfaces. The rest of the lumen is filled by a pink staining concentrically laminated amorphous material. The lumina in the epididymis are lined by tall columnar cells and contain masses of spermia. No areas of

FIG. 4. Frater's case. Cyst completely invested by tunica albuginea. (× about 50.)

caseation necrosis or giant cells are noted anywhere in the preparation.

"Pathological diagnosis: Cyst of the testicle."

The finding of a rare condition has always been subjected to considerable speculation as to its etiology. Various hypotheses have been advanced in regard to the etiology of these cysts. Some believe them to be retention cysts. Barach believes trauma to be an etiological factor. Smith believed gonorrhea which secondarily infected the epididymis and vas deferens caused an occlusion resulting in the cystic tumor. The possibility of their being congenital, arising from the remnants of the Mullerian and Wolffian ducts, has been advanced.

We have no etiological factor to suggest in our case. There is no history of gonorrhea, injury or abnormality of the testicle.

SUMMARY

1. A cyst of the testicle is reported.

2. The differential diagnosis of cyst of the tunica albuginea and cyst of the testicle can only be made by microscope study.

3. There are no diagnostic features of this disease, either subjective or objective. So far we have no means of differentiating an early malignant tumor and this benign disease.

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DISCUSSION

Dr. F. H. Colby: Dr. Jenkins said there is no means of making a differential diagnosis between cyst and tumor. The Prolan test is said to aid in differentiating the benign and malignant tumors of the testicle.

Dr. Richard Chute: In regard to Dr. Colby's remarks about the Prolan test, Dr. Smith and I are using this in the Tumor Clinic at the Massachusetts General Hospital and I would like to ask Dr. Smith to express his present opinion of the Prolan test.

Dr. G. G. Smith: I think Dr. Chute refers to one case we had, a case I operated on when the College of Surgeons was here, in which the Prolan test was negative but the man proved to have a carcinoma of the testicle, so I feel that the fact that the test is negative doesn't necessarily exclude tumor.

Dr. Ralph H. Jenkins: As to whether the cyst had anything to do with this patient's unusual sexual capacity, I do not know. The question as to whether the quantity of excretion of Prolan A would be a factor in distinguishing between malignant and benign tumors of this kind should be considered. This particular case was treated before this test was given much consideration.

Dr. Clyde L. Deming: I would like to say that this man has proved to be much better with one testicle than with two. This case appeared before the Prolan A test was used for diagnostic purposes so we do not know much about what effect the test might have in an individual of this type. That is why Dr. Jenkins is hedging a little bit, because it is a case of three or four years' standing, before we knew much about Prolan A test. Had the test been recently applied, we would undoubtedly have been able to answer the question which has been asked.

NATIONWIDE STUDY OF FIREWORKS ACCIDENTS

A nationwide study of the nature, causes and results of fireworks accidents was inaugurated July 5 by the American Museum of Safety with the coöperation of public health authorities and safety organizations throughout the country, it is announced by Arthur Williams, President of the Museum.

The study will seek to ascertain how serious a hazard to life, limb and sight fireworks are, which particular items of fireworks are involved in most accidents, what influence—if any—prohibitory legislation has on the frequency of fireworks accidents, and what are the sources of the fireworks causing accidents.

The study will be under the direction of Dr. Leland E. Cofer, internationally known authority in the fields of public and industrial health, former Assistant Surgeon General of the United States Public Health Service, and former Director of Industrial Hygiene for the State of New York.

Dr. Cofer will be assisted by the following representatives of other national agencies concerned, all of whom have had long acquaintance with the problem of fireworks accidents and who now constitute the Fireworks Accident Prevention Committee of the Museum of Safety:

Dr. Donald B. Armstrong, Vice-President, Metropolitan Life Insurance Company; Lewis H. Carris, Managing Director, National Society for the Prevention of Blindness; C. H. Fleming, Executive Secretary, Pyrotechnic Industries, Inc.; James L. Fri, Managing Director, Toy Manufacturers of the U. S. A.; Lee F. Hanmer, Director, Recreation Department, Russell Sage Foundation; Albert S. Regula, Secretary, American Museum of Safety; Louis Resnick, Director of Industrial Relations, National Society for the Prevention of Blindness; James Speyer, Treasurer, American Museum of Safety; W. S. Topping, Assistant Chief Inspector, U. S. Bureau of Explosives; and Franklin H. Wentworth, Managing Director, National Fire Protection Association.

Announcing this study, Mr. Williams said, "For years there have been seriously conflicting statements and notions as to the consequences of using fireworks to celebrate Independence Day. The country has been told that we now have a sane and safe Fourth, and there are many evidences bearing out this claim. On the other hand, we are told by individuals and organizations who have made admittedly casual investigations that use of fireworks in connection with the Fourth of July is still hazardous and that it results in much loss of life and serious injuries to thousands of persons, principally children.

"This year, for the first time, we should learn without any question of doubt just how serious the fireworks hazard is." — *Bulletin of the American Museum of Safety.*

EDWIN KLEBS*

A Centennial Note

BY LEONA BAUMGARTNER, M.D.†

I. LIFE

EDWIN Klebs, whose hundredth birthday fell on February 6, 1934, was one of the pioneers of bacteriology and he also made distinguished contributions in other fields of medicine. It therefore seems appropriate, at this time, to recall some of his achievements. The name of Klebs is irrevocably attached to the bacillus he first described in 1883 as found in the throats of patients with the then undifferentiated group of conditions called "croup"[26]. Less well known are many other contributions of almost equal significance. Thus, he described acromegaly[6] before Pierre Marie; fifteen years before Metchnikoff he inoculated primates with the virus of syphilis[11, 22, 23]; he[15] described the isolation of colonies of bacteria on solid media before Koch; he was the first to produce tuberculosis experimentally in animals by the injection of milk from infected cows, thereby establishing the bovine origin of the disease[17]; and he described typhoid bacilli and attributed a causal relation between the organisms and the disease before Eberth, who is generally accredited with their discovery. The establishment of priority *per se*, however, can seldom be a matter of as great concern as the estimation of the influence exerted by a man upon the history of a given subject, and it is from this point of view that we propose to take up the work of Edwin Klebs.

Theodore Albrecht Edwin Klebs was born February 6, 1834, in the decade of the nineteenth century which saw the birth of Johannes Müller's "Physiologie", Cruveilhier's school of pathological anatomy, and the cell theory of Schleiden and Schwann. It is perhaps not surprising, therefore, that as he grew to manhood and finished school in his native Königsberg, he turned to medicine instead of to the law to which the fortunes and traditions of his family had long been bound. Rathke, Helmholtz, Burdach, and Werther were his first teachers during five semesters in Königsberg. At twenty-one years of age he enrolled for further study at Würzburg under the then popular and stimulating medical faculty composed of Virchow, Kölliker, Scherer, Bamberger, Leydig and Scanzoni. In later years Klebs often spoke of these days in glowing terms. Virchow impressed him so deeply that at the first opportunity he followed him to Berlin as an assistant. This was in 1861, three years after the "Cellularpathologie" had appeared and after Klebs had completed his doctorate. A brilliant group was then gathered in the laboratories of Virchow's newly erected Pathological Institute: Edwin Klebs, Oskar Liebreich, Julius Cohnheim, Friedrich von Recklinghausen, Willy Kühne, and Ernst Leyden, men who were unconsciously fitting themselves to occupy in later years the chief academic posts in the German-speaking medical world, and who already had commenced the researches which were to give to the school the credit of formulating the theories which dominated pathology for the next half century.

After these preparatory years Klebs went first to Berne (1866), then as Recklinghausen's successor to Würzburg (1872), and the following year to Prague at Heering's request, returning to Zürich, Switzerland, in 1882. The six years at Berne he described as the happiest of his life, but the nine at Prague were the most fruitful scientifically. He not only did original work of first importance in bacteriology, but in addition he taught many young men, and developed three important medical journals. Among his pupils in these academic years were numbered Ernst Ziegler, Zahn, Hanau, Sazka, Köster, Fischl, von Jaksch, Eppinger, and Lubarsch. It was the day of the founding of the great German scientific journals, journals so intimately bound up with their individualistic founders as to bear their names (Virchow's, Ziegler's, Pflügler's, etc.). The three journals which Klebs himself inaugurated were named in a style more approved by a modern scientific world which does not so readily countenance great individualism: *Die Prager medizinische Wochenschrift* begun in 1876, the *Correspondenz-Blatt für Schweizer Aertzte* appearing in 1871, and the *Archiv für experimentelle Pathologie und Pharmakologie* founded with Naunyn and Schmiedeberg in 1873. All have withstood the rigors of passing fashions in scientific publishing and have served a useful purpose for their respective readers.

Upon leaving Zürich, where he had worked for eleven years, Klebs went in 1893 to Carlsruhe for a year, then came to America, settling (1895-96) in the laboratory of a private tuberculosis sanatorium in Asheville, North Carolina, and in August of 1896 moving to Chicago as professor of pathology in Rush Medical College. But in 1900 he returned to Europe to live successively in Hanover (where the *Kausale Therapie*, the fourth and only short-lived medical journal edited by Klebs, was started), Berlin, Lausanne, and Berne. The many vicissitudes of his life, so often rendered poignant and even dramatic by the changeable temperament of the man, have been adequately commented upon by his

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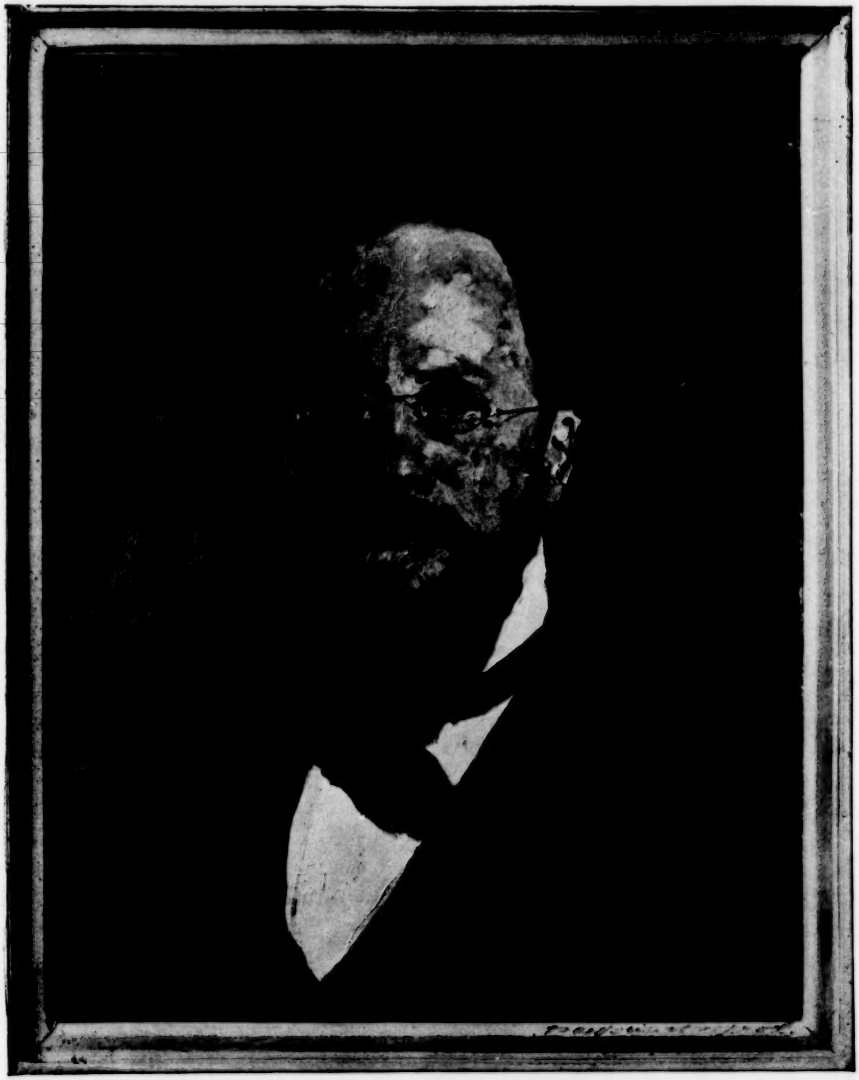
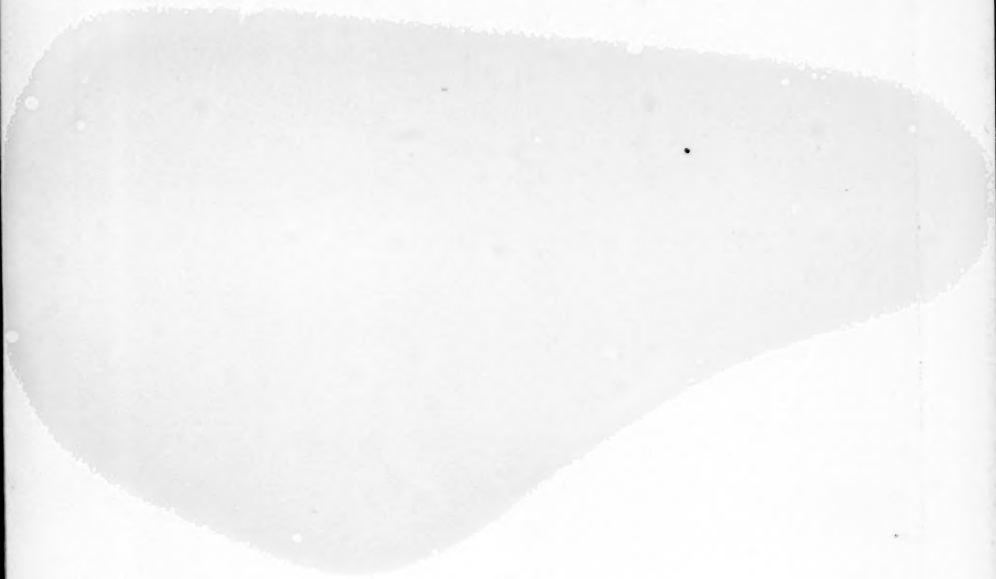


Portrait of Dr. Edwin Klebs, painted in Rome, 1879, signed *H. Treuenfels a. I. Prof. Klebs. Rom 79*, reproduced through the courtesy of Dr. Arnold C. Klebs.



various necrologists[1, 2, 3, 4, 5, 7, 8, 9, 35, 37, 38], but his work gives little or no evidence of restlessness or indecision of thought.

II. WORK

His early studies on the bacteriology of gunshot wounds[15, 16], growing out of his experiences in 1870 and 1871 in military hospitals in Carlsruhe and antedating by one year Koch's great memoir on the subject, had convinced Klebs of the bacterial nature of wound infection. He immediately devoted himself to impressing the ardent followers of the Virchow school of the importance of these external agents. These antagonists of humoral pathology, engrossed in the myriad of fresh vistas which new histological methods were bringing beneath their microscopes (Klebs' paraffin-embedding[13], Cohnheim's frozen sections, etc.), found it difficult to accept the bacterial theory of infection and to Edwin Klebs must be given a large share of the credit for winning them over to this view. His own bacteriological studies included a treatise on the cause of anthrax[19] appearing a year after Koch's work, the first description of the bacillus of typhoid fever[24, 25] and of diphtheria[26], and extended studies on cholera[27, 28, 31] and malaria[30, 33, 34]. The use of solid media in the form of sturgeon's glue was fully described by Klebs in 1872[15], nine years before Koch. But Klebs saw in the method not a means of separating bacterial species, as did Koch; he was interested in the different colonies as different stages in the growth of the same organism, an interesting observation in view of the recent ideas concerning bacterial dissociation. He felt he could secure pure cultures with his method of fractional dilution, i.e., killing off the contaminants in an impure culture by successive transfers through a series of fresh media. The Koch postulates, first enunciated in 1882, had certainly been foreshadowed in Klebs' statements made in 1877 concerning the etiology of tuberculosis[20]. Having discussed the anatomy of the local lesion, he pertinently remarks:

"The simplest way to prove that the cells of the diseased animal neither produce nor in any way increase the virus of tuberculosis is to cultivate it outside of the organism, under conditions which do not destroy the life processes of the cells of the warm-blooded hosts. If, under such conditions, there is still an increase in the toxic action, this must be dependent upon the presence of tissues of the animal body, remains of dead tissue cells, or upon components of the inoculum which, usually foreign to the animal, developed within the tubercle." (a)

(a) "Der einfachste Weg, um zu beweisen, dass nicht die Zellen des erkrankten Tieres den Giftstoff der Tuberculose produciren oder zu seiner Vermehrung irgendwie beitragen, besteht in der Cultivirung dieses letzteren ausserhalb des Organismus, unter Bedingungen, welche die Lebensfähigkeit der Zellen des Warmblüters vernichten. Findet unter solchen Bedingungen dennoch eine Zunahme der giftigen Wirkung statt, so kann dieses nur entweder von den noch vorhandenen Bestandtheilen des Thierkörpers, den Resten der abgestorbenen Zellen desselben abhängen oder von Bestandtheilen des Impfmaterials, welche, dem Thierkörper ursprünglich fremd, sich innerhalb der Tuberkel entwickelt haben."

Having described cultivation experiments, using fresh egg white as a culture medium, he continues:

"If it can thus be established, as the older inoculation experiments indicated and the ones presented today corroborate, that tuberculous infection can not only be induced by inoculation of tuberculous tissues but also with organisms which are found in such tissues and can be grown outside the body, it becomes necessary to establish the presence of these organisms in the diseased tissues from human and animal tuberculosis." (b)

And in proof of such a proposition he describes at length the organisms he has seen in tubercles. The final step of animal inoculation with culture virus is concisely outlined:

"Small amounts of the products of this cultivation experiment inoculated into the peritoneum of healthy animals, result in the development of a miliary tuberculosis exactly as does the implantation of tuberculous tissue in the same locus." (c)

It is of more than passing interest that in the discussion which followed the reading of the paper no comment was aroused over these experimental results for it is evident that they include the salient points of the well-known Koch postulates upon which the recognition of any organism as the etiological agent of a given clinical entity has subsequently been based.

Klebs continued to be absorbed in the problem of tuberculosis during his entire life. His inaugural dissertation and first published work had been entitled *De mutationibus quae in intestino inveniuntur tuberculosis*[12] and he wrote over seventy individual articles[10], largely concerning the therapy of the disease. The one of greatest interest at present, in view of the recent BCG vaccination method, is that description of the use of attenuated strains of tubercle bacilli from cold-blooded animals, in the development of active immunity in guinea-pigs and human beings[32]. His experimental production of the disease with milk of bovine origin has already been noted. (d)

His many contributions to the field of path-

(b) "Wenn hierdurch auch die Thatsache wohl zweifellos festgestellt ist, dass tuberculöse Affectionen nicht allein, wie die älteren Impfversuch lehren und die heute vorgelegten Präparate von Neuem bestätigen, durch Uebertragung von tuberculösen Geweben, sondern auch durch Organismen geschehen kann, welche aus solchen Geweben stammen und ausserhalb des Körpers sich vermehrem können, so fordert diese Erfahrung doch wiederum auf, den Nachweis dieser Organismen in dem erkrankten Gewebe bei genuiner Tuberculose der Menschen und Thiere zu versuchen."

(c) "Geringe Mengen von den Producten dieser Culturversuche, in die Bauchhöhle von gesunden Thieren eingespritzt, führten ebenso zur Erzeugung von miliarer Tuberculose des Peritoneums, wie dieses der Fall ist, wenn man genuine Tuberculose an dem gleichen Orte implantiert."

(d) It is unnecessary to include all of Edwin Klebs' printed works in view of the excellent bibliography of his scientific writings prepared by his son, Arnold C. Klebs[36], which, with its convenient classification of titles according to their contents, i.e., (1) normal and pathological anatomy and physiology, (2) pathological etiology, (3) prophylaxis and therapeutics, (4) historical material and reviews, (5) texts, and (6) periodicals, contains about two hundred and twenty-five titles. We were able to locate only a very few additional items, and only one of any importance[3].

ological anatomy and physiology included the first experimental production of valvular disease of the heart[18] and the recognition of the bacterial infection in the production of the subsequent endocarditis[21], his description of hemorrhagic pancreatitis as a rapidly fatal disease[14], the introduction of the paraffin embedding method[13], and his monumental texts, *Handbuch der pathologischen Anatomie*[14] (1869-1876) and *Die allgemeine Pathologie, oder die Lehre von den Ursachen und dem Wesen der Krankheitsprocesse*[20] (1887-1889) which, though never completed, became invaluable source books for pathologists for the next twenty-five years. To the general understanding of the processes of cell necrosis with its phases of karyolysis and karyorrhexis, of infarction, thrombosis and embolism he added much that was illuminating.

It is commonly conceded that Pierre Marie first described the clinical entity acromegaly which is still recognized by the name he so aptly coined. Of Klebs' several biographers[2, 4] only Garrison[7] draws attention to the fact that Klebs' paper on gigantism[9] furnishes a complete and excellent description of the same disease, which antedates that of Pierre Marie by two years. Marie's classical description, appearing in the *Revue de Medicine* in 1886[36], was concerned with the clinical aspects of the two cases of the disease he had seen simultaneously in Charcot's clinic, with notes on five apparently similar cases which he collected from the literature. No suggestion was made of the relationship of the disease to the pituitary gland, neither case having come to autopsy at this time. Marie was content to speculate on the possible etiology of the disease, considering such widely divergent explanations as an involvement of the sympathetic nervous system, a generalized disease simulating rheumatism, or a developmental defect. Klebs and his clinical collaborator, Frit[z]sche, describe in detail one case, Klebs being responsible for the painstaking and excellent postmortem descriptions, which in themselves are models of the careful work produced by the well-trained pathologist of the nineteenth century. Moreover, armed with the pathological findings Klebs proceeded to explain the etiology of the condition in broad physiological terms of growth and development, concluding that a generalized process of hypertrophy was at work, the pituitary hyperplasia being a manifestation of that process and the thymus being definitely involved. It is also interesting to see that he was the first to recognize that pituitary tumors had been associated with similar cases reported in the literature, a point overlooked by Pierre Marie.

III. CHARACTER AND INFLUENCE

That Klebs did not push many of his investigations to what to us seems their logical conclusion becomes apparent as soon as one surveys his work. This was a natural failing of early pioneers. They moved in many entirely new fields, and they moved rapidly from one to the other. Klebs described the typhoid bacillus, but it was left for Gaffky to cultivate it four years later. The work on toxicity and non-infectivity of bacterial filtrates[15, 16] was clearly of great significance, but it was not until years later that von Behring, Roux, and Yersin were to clarify the action of toxins and antitoxins. Some of these apparent shortcomings may well have been the result of his restless nature, which always interested itself in new aspects of a problem and was often impatient of the necessities of the moment. Several anecdotes give evidence of these tendencies. Dr. Welch repeatedly told a characteristic story illustrating this trait in Edwin Klebs. On the occasion of a visit by Klebs to Welch's laboratory at Johns Hopkins, Welch placed under several microscopes a series of preparations from a case of Landry's paralysis in which some of the thrombotic changes Klebs had first described were beautifully demonstrated. Welch said that he found Klebs looking into one after the other with but little comment. When pressed for some expression, he replied, "Evidently very good preparations, but those thrombi do not interest me any more. As I was looking at these cells I realized how much we have still to learn about the entire mesoderm." And at least two of Klebs' recorded statements give evidence that he recognized his own tendencies. One is a direct reply to a criticism hurled at his first announcement of the discovery of the diphtheria bacillus at the Second Congress for Internal Medicine held in Wiesbaden on April 19, 1883. To Dr. Körte of Berlin, who suggested that the question of etiology could be settled only after the organisms had been isolated in pure culture and then successfully inoculated into animals, Klebs replied (Reference: p. 172 of No. 26) "I admit that I have devoted my efforts to this task for years, but gentlemen, he who works a great deal has many failures and, after any one of us has worked twenty-five years in this field, he cannot be blamed if he does not successfully achieve the desired goal." In later years, however, he is reported by one of his students to have said, "I was the first to see and partially describe many pathogenic bacteria, but I had no success with those methods which yielded conclusive evidence. And everything depends upon these methods, a basic idea in modern scientific research. Never fail to search for new methods[3]." And it is as a pathfinder, always searching for the new and the unknown, that Edwin Klebs was known to his contemporaries and deserves to be remembered.

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MEDICAL PROGRESS

PROGRESS IN PSYCHIATRY FOR 1934

BY KARL M. BOWMAN, M.D.*

DURING the past year there has been continued work in formulating a plan to provide some type of certification for qualified psychiatrists. This has finally resulted in combined action by The American Psychiatric Association, The American Neurological Association, and The Section on Neurology and Psychiatry of the American Medical Association. As a result a meeting was held in New York on October 20, 1934 at which the American Board of Psychiatry and Neurology, Inc., was organized. The following officers were elected:

Dr. H. Douglas Singer, President.
Dr. C. Macfie Campbell, Vice President.
Dr. Walter Freeman, Secretary-Treasurer.

Other Members of the Board are the following: Dr. Lewis J. Pollack, Dr. George W. Hall, Dr. Franklin G. Ebaugh, Dr. Lloyd H. Ziegler, Dr. J. Allen Jackson, and Dr. Adolf Meyer. This Board will have authority to issue a certificate indicating that the individual is a qualified psychiatrist or neurologist or both. Psychiatry and neurology have by this step set up certain standards for a qualification and are following the example of a number of the other specialists in working out a plan to prevent unqualified persons posing as specialists in a particular field.

The annual meeting of The American Psychiatric Association was held at New York City, May 29-June 1, 1934. Prior to this meeting the National Committee for Mental Hygiene again arranged a two-day conference on "The Teaching of Psychiatry in Medical Schools." This conference again stressed the importance of psychiatry in the curricula of the medical schools and considered plans for improving such teaching.

There were apparently two points of view which showed considerable divergence.

One group felt that the teaching of psychiatry to medical students should be largely through the use of general medical and surgical material in which the psychological factors of physical disease were stressed. Certain members of this

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group even went so far as to hold that no formal teaching of the psychoses should be given.

The other point of view was that since over half of the hospital beds in this country were filled with cases of mental disease, anyone going into the practice of medicine needed some acquaintance with these serious mental disorders. It was pointed out that mental disorders are most commonly seen first by the general practitioner and that he therefore needs to have some knowledge of this subject. Furthermore, it was held that the general public turned to the physician for understanding and advice on the problem of mental disease, and if the physician who was graduated from a Class A medical school has no more knowledge than the laity as to the problems of mental disease, it would cause the lay individual to lose faith in the physician and to turn more to unqualified practitioners in the field of mental disease.

In line with this a report was submitted by Dr. Franklin G. Ebaugh, entitled "Ideal Standards for the Teaching of Psychiatry in Class A Medical Schools" which was endorsed both by the Advisory Committee on Psychiatric Education of the National Committee for Mental Hygiene and by the Committee on Psychiatric Education of The American Psychiatric Association. It was held that psychiatry should be one of the fundamentals of the basic training of every physician and wherever possible, instruction should be given during all four years. The ideal type of curriculum was held to consist of:

Sixteen hours of "pre-clinical instruction in psycho-biology during the first year."

Sixteen hours of "psycho-pathology and methods of study and formulation" in the second year.

Sixty hours of "clinical instruction, didactic work, clinics, and clerkships" in the third year.

Sixty hours of "out patient work, inter-departmental consultation participation, and examination of individual cases" during the fourth year.

On June 21, 1934 Yale University conferred the honorary degree of Doctor of Science on Dr. Adolf Meyer as an expression of the great pioneer work which he has done in American Psychiatry.

The second series of the Thomas William Salmon Memorial Lectures was given by Dr. C. Macfie Campbell at the New York Academy of Medicine on April 13, 20 and 27, 1934; his subject was "Trends in Psychiatry." These lectures are to be published by the Thomas William Salmon Memorial.

In June, 1934, Dr. James V. May resigned as Commissioner of Mental Diseases in Massachusetts and resumed his former position as Superintendent of the Boston State Hospital. Dr. May had agreed to accept the Commissionership for a period of one year following the death of Dr. Kline, but had insisted that he should be free to return to his former position at the end of that period. Dr. Winfred Overholser who had for many years been Assistant Commissioner was appointed Commissioner of Mental Diseases by Governor Ely.

The Rockefeller Foundation made a substantial gift for the increase of work in psychiatry at the Harvard Medical School. The bulk of this fund was given for the establishment and maintenance of a psychiatric unit at the Massachusetts General Hospital to be under the direction of Dr. Stanley Cobb and part to establish some new work at the Boston State Hospital.

It cannot be said that any new or startling discoveries have been brought forth in the field of psychiatry during the past year. The literature has continued to be quite extensive and prolific and all varieties of topics have been dealt with.

In the field of heredity, further studies of the factors in dementia praecox and manic depressive psychoses made by Pollack and his associates in New York State and published in the Psychiatric Quarterly indicate the importance of the hereditary factor in both of these diseases.

Two recent articles by E. Bleuler of Zürich defend the idea that certain acquired mental characteristics can be inherited. This is, of course, contrary to the viewpoint of most biologists, but coming from the pen of such a distinguished physician the articles merit serious consideration. It is perhaps worth calling attention to the fact that many prominent men in the field of psychiatry, particularly a number in the psychoanalytic field, have insisted on an inheritance of acquired mental characteristics.

There has also been further work concerning the inheritance of feeblemindedness which also confirms the hereditary nature of this condition where injury and disease have been ruled out as etiological factors. One of these articles suggests that feeblemindedness may be transmitted as a recessive trait.

Various attempts have been made to work out causative factors in mental disease particularly in schizophrenia. Most of these studies have led to negative conclusions. Thus, Freeman and Looney at the Worcester State Hospital attempted to determine whether a toxin was present in the blood or urine of schizophrenic patients. The question of toxicity was determined by noting the effect of dilutions of blood and urine of the patients on the growth of Lupinus Albus. The results were negative and showed no evidence of any such toxin.

Rosanoff and Inman-Kane after a study of premature birth and underweight at birth conclude that children born prematurely and underweight are much more likely to sustain cerebral injury at birth and hence this condition is a factor in the production of mental deficiency.

A study of the etiology of Mongolism with

special reference to its occurrence in twins by Rosanoff and Handy emphasizes the idea that the most important factor appears to be the age of the mother and the authors feel that some disease of the ovary which occurs with the advancing age of the mother is of the greatest importance in producing this condition. They point out that the condition is more frequent in boys than girls and discuss further factors such as the possibility of the spermatozoon exerting a protective influence.

One of the most pretentious and important articles concerning the nature of mental disease is by Pavlov entitled "An Attempt at Physiological Interpretation of some Neuroses and Psychoses." This article appears in the *Journal of Mental Science* for April, 1934. It can hardly be presented intelligently in brief form and the reader is referred to the article. It may be stated, however, that Pavlov found that by using his concept of the conditioned reflex he was able to work out a characteristic mental state which he called "pathological inertness", and which he felt explained some of the conditions found in mental disease particularly in the obsessional neuroses and in paranoia.

In the field of treatment there have been many articles published but few that seem of much significance. Many have to do with methods of treatments which have already been tried out to some extent but a few represent some new method. Further use of methods of prolonged narcosis have brought out little that is new. Some authors call attention to the need of psychotherapy as a part of any such plan; while there are others who feel that most of the results achieved are due to the psychotherapy rather than to the prolonged narcosis. The standards for determining cure or improvement are so difficult to establish in psychiatry and the lack of control material is so common that most articles on the subject of treatment are largely impressionistic. This is particularly true concerning the manic depressive psychoses where the patients ordinarily recover if no treatment at all is given. When one reads, therefore, of 37.7 per cent of cases of manic depressive psychoses recovering under prolonged narcosis treatment, there seems little value to such figures.

One interesting result of the increased frequency of encephalograms is the report from several sources of total improvement in certain cases as a result of the injection of air, where certain cases of head trauma appear to have been relieved of headache, dizziness, and even convulsive attacks. Further study along this line seems indicated and it may be that some important therapeutic results can be achieved by such a method of treatment or by some modification of such treatment.

In recent years the concept of suboxidation of the brain as a cause of dementia praecox has attracted considerable attention. Mott held strongly to this view and it is now generally accepted that there is a tendency toward a low basal metabolism in dementia praecox. At the New York Psychiatric Institute an attempt was made to treat five cases of dementia praecox by keeping them continuously for two and a half months in an oxygen chamber of approximately fifty per cent oxygen. During this period the patients were also given further treatment with carbon dioxide and oxygen. Psychotherapy was also a part of the treatment. Three control groups were used, one group receiving the continuous oxygen treatment and the combined carbon dioxide side treatment, but no psychotherapy. A second group simply lived in the fifty per cent oxygen atmosphere. A third group lived outside the oxygen chamber but received the daily treatments of carbon dioxide and oxygen. From the standpoint of therapy there seemed little value to the oxygen treatment. The cases that received psychotherapy as well did best.

Various other chemical treatments have been attempted and treatment has been carried out by injections of placental blood, of sulphur and gold, of insulin, and of manganese, also various endocrine preparations and extracts of nervous tissue have been used. The results obtained do not point to any important new method of therapy.

In the treatment of depressions hematoporphyrin has apparently had some value and it is probable that within the next few years there will be further experiments with this drug. There also has been further use of ovarian extract in the treatment of involutional melancholia with some rather optimistic reports.

There has been further use of fever therapy in the treatment of general paresis and it now seems firmly established that by the use of fever therapy, particularly malaria, complete remissions may be obtained in as high as one-third of all cases treated and improvement in another third. Besides malaria a large number of other methods have been used to produce hyperpyrexia. Diathermy continues to be used although the danger of burns is a serious drawback. The injection of substances, such as typhoid vaccine, the use of the electric blanket, and the hot water bath, all seem to be possible methods which are somewhat simpler. Still more recently an air-conditioned cabinet has been used. It is still too early to determine the advantages or disadvantages from it.

There has also been some excellent work in treating hyperthyroid children with thyroid preparations and following the intellectual development of the child by very careful psychological tests. These tests appear to demonstrate the value of thyroid treatment and indicate that the earlier a condition is recognized and treatment instituted, the greater will be the intellectual development.

An interesting study on blood sugar in relation to emotional reactions by Whitehorn confirms the findings of previous writers that there is little relation between the fasting blood sugar and the emotional state of the psychotic patient. The vast majority of such patients, even in states of extreme excitement, show a normal blood sugar. Where hyperglycemia occurred it was usually in some organic type of case or in women at about the involutional period of life.

An excellent article by Paul Schilder appears in *The American Journal of Psychiatry* for July, 1934. The article, entitled "Psychic Disturbances After Head Injuries" does not lend itself to a brief summary and is a discussion of the various types of mental symptoms occurring after brain injury.

The great increase in the smoking of marihuana cigarettes has led to several studies of this subject. The habit has apparently spread from Mexico and Latin-American countries and the drug itself is the American form of hashish or Cannabis Indica. There have been many reports of the effects of smoking these cigarettes and the habit seems particularly prevalent among high school students. A certain exhilaration occurs, a tendency to laugh easily, oftentimes an alteration in the sense of time and space, and repressions, particularly sexual, are removed. There is no tendency to habit formation as in opium or cocaine. The drug is little used by criminals who commonly prefer opium or cocaine. A very excellent summary of the subject, entitled "Marihuana Intoxication; A Clinical Study of Cannabis Sativa Intoxication" by Bromberg appears in *The American Journal of Psychiatry* for September, 1934.

A number of books have appeared which are of considerable interest to the psychiatrist and mention may be made of a few which could probably be of value to the general medical profession.

Elton Mayo has written on "The Human Problems of an Industrial Civilization" in a manner which should interest everyone.

Huddleson's "Accidents, Neuroses and Compensation" gives an excellent summary of our present knowledge of this troublesome condition.

Weyhoefen's "Insanity as a Defense in Criminal Law" has attracted considerable attention and has been generally accepted as one of the best books on this subject.

Nathan G. Goodman has written a book on "Benjamin Rush, Physician and Citizen" which should interest every follower of medicine. The discussion of Rush's service during the Revolutionary War, his criticisms, and quarrel with General Washington, and his ideas concerning mental disease all afford interesting reading.

Campbell's "Human Personality and the Environment" which represents the substance of six lectures given at the Lowell Institute is a book with which all students of personality should become familiar.

Stefan Zweig's "Mental Healers—Franz Anton Mesmer, Mary Baker Eddy, Sigmund Freud" is a very readable account of these three individuals.

The last volume of *The Association for Research in Nervous and Mental Disease*, entitled "The Biology of the Individual" is also a study of personality which will remain for some time as an important contribution to this subject.

AN ADVERTISED HEALTH CLINIC

A pamphlet has been circulated in which appears an advertisement of a Health Clinic in Boston with a doctor and dentist in attendance.

The doctor's name does not appear in the Directory of the American Medical Association so far as we have discovered, and the Massachusetts State Board of Registration in Medicine also reports that this doctor's name does not appear in the list of registered physicians. The advertisement sets forth that complete physical examination will be made for $1.00.

Perhaps this may be a plan for emulating the practice of the Canadian doctor who charged $1.00 per treatment and has been reported to be making a financial success of manipulation of the feet of his patients.

Whether this venture will be a financial success is doubtful, because the public is growing wary of advertised medical service although there is a general impression that adequate advertising of most anything will bring favorable returns.

If it is found, on further investigation, that this doctor is not registered, there will be more doubt of his competing successfully with other practitioners in Boston. There seem to be some obstacles in the way of this clinic.

THE FLOATING CONGRESS

About three hundred physicians from the United States, some with their wives, sailed on the Queen of Bermuda for Rio de Janeiro June 30, as participants in the Floating Congress.

The medical activities will be divided into seventeen sections of medical discussions. The Brazilian medical profession has arranged scientific sessions at Rio de Janeiro and Sao Paulo with special exhibits at the Oswaldo Cruz Institute of Tropical Medicine and at the Sao Paulo University with its Institute Sero-Therepeuttico.

CASE RECORDS *of the* MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 21281

PRESENTATION OF CASE

First Admission. A seventy-seven year old unemployed American entered complaining of swelling of his neck.

Ten months before entry he began to feel weak and complained of moderate shortness of breath on exertion. His physician believed he had influenza and kept him in bed for about a week. Since then he continued to feel weak and tired very easily. One month later he noticed a swollen painless gland in the right side of his neck. The gland was not red, hot or tender. Six months ago the swelling gradually increased in size and a similar swelling appeared on the left side of the neck. Four months before entry he began to have drenching night sweats which had continued. Five weeks before entry he visited another physician who found enlargement of the axillary and inguinal glands and gave him a course of intravenous treatments, probably sodium cacodylate. He lost about five pounds during this illness.

The family, marital and past histories are non-contributory.

Physical examination showed a well-developed and nourished, slightly pale man. The pupils were slightly irregular. In the right neck there were large, firm, matted, very slightly movable glands measuring from 1.5 to 2.5 centimeters in diameter. There were similar enlarged glands on the other side many of which, however, were discrete. The glands in the axillae and groins were similarly enlarged. One in the right epitrochlear region measured one centimeter in diameter. All the glands were firm and nontender. The lungs were clear. The heart was not enlarged. The sounds were indistinct and distant. The blood pressure was 100/70. The abdomen was held very tense and examination was unsatisfactory. The prostate was enlarged, firm, but not hard.

The temperature was 99°, the pulse 78. The respirations were 26.

Examination of the urine was negative. The blood showed a red cell count of 4,400,000, with a hemoglobin of 70 per cent. The white cell count was 5,400. The differential smear showed 46 per cent polymorphonuclears, 36 lymphocytes, 3 large mononuclears, 5 eosinophils, 9 lymphoblasts and 1 unclassified. The red cells showed moderate achromia. The platelets were normal. Red blood cell counts on the sixth and fourteenth days were 2,550,000 and 2,280,000 respectively. The hemoglobin was 60 per cent. The stools were negative. A Hinton test was negative. The phenolsulphonephthalein test showed 90 per cent excretion at the end of one hour. The nonprotein nitrogen was 34 milligrams per cent.

An x-ray examination of the chest was negative.

On the fifth day he developed a skin eruption which consisted of reddened, slightly raised, oval lesions. These occurred for the most part on the arms, legs and buttocks. Three days later the eruption had become widespread. A skin consultant believed the lesion was a toxic erythema, probably due to a drug. A few days' treatment with a wash consisting of phenol and chloral hydrate gave considerable symptomatic relief. A biopsy of one of the axillary glands showed lymphoblastoma, sarcomatous type. X-ray treatment was begun and he was discharged one month after admission, the glands having disappeared.

Second Admission, five months later.

Following discharge he improved considerably and was able to work quite strenuously. However, after a few weeks he began to have shortness of breath and weakness. He reported to the x-ray department for treatment. One month before admission while being observed in the x-ray clinic he became very tired and could hardly get home. For the next two days he had some nausea and vomiting. During the month before entry his tongue became cracked and burned especially when he ate sweets. He entered this time because of marked weakness, fatigability and dyspnea.

Physical examination showed a thin, tired-looking, slightly jaundiced man. The hair was very scant in the axilla and groins. The tongue was smooth. There were a few moist râles at the bases. The blood pressure was 100/60. The liver was felt two fingerbreadths below the costal margin. The spleen was felt just at the costal border. There was slight edema of the ankles. There were a few smooth preauricular glands on the right and similar ones in the axillae and groins.

The temperature was 98.4°, the pulse 90. The respirations were 20.

Examination of the urine was negative. The blood showed a red cell count of 1,110,000, with a hemoglobin of 50 per cent. The white cell count was 3,250, with a differential of 54 per cent polymorphonuclears, 36 lymphocytes, 1 eosinophil, 1 basophil and 8 immature cells. There was marked variation in the size and shape

of the red blood cells. The platelets were normal. There was an occasional reticulocyte. A gastric analysis showed no free hydrochloric acid even after ergamin. The icteric index was 8.

He was first treated with a highly purified liver extract which produced no apparent response. Two weeks after admission intensive parenteral liver treatment was started following which there was an excellent response. There was marked improvement in strength and appetite and almost complete disappearance of the soreness of his tongue. His red blood cell count was 2,070,000, with 26 per cent reticulocytes. About one month after entry he received x-ray treatment over both upper cervical and preauricular regions. The glands became swollen and somewhat softer following this treatment. He was discharged improved during the sixth week and was told to continue with the liver treatment.

Third Admission, four months later.

During the interval he was seen in the x-ray clinic. His red cell count had remained around 4,000,000. A few weeks before this entry he developed headaches and blurring of vision. Several of the glands reappeared. On the day before admission, after an x-ray treatment over the skull to include the eyes because of the enlarged glands above each orbit, he became very weak and developed a severe headache. His vision failed.

On physical examination prominent subconjunctival hemorrhages were found. There were soft indefinite masses over each orbit. The liver and spleen were enlarged to percussion but were not felt. The glands in the neck, groin, elbow, axillae and back of skull were enlarged to one centimeter in diameter.

Examination of the blood showed a red cell count of 3,270,000, with a hemoglobin of 60 per cent. The white cell count was 5,200, 57 per cent polymorphonuclears and 10 per cent immature forms.

Liver by mouth and liver extract intramuscularly were given. He was given another course of x-ray treatment but he developed signs of collapse and pneumonia and died six weeks after admission.

Differential Diagnosis

Dr. Wyman Richardson: I hate to see a patient with swelling of the neck due to glands because I am always wrong in making a diagnosis of the cause of the swelling, at least until biopsy reports are available. Fortunately in this case they are available.

We have a history of enlarged glands and the only thing that seems to me to be important is that he has had drenching night sweats. I feel that that is rather against the diagnosis of metastatic carcinoma and more in favor of some lymphoid tumor. As to the intravenous treatment, I am not sure what was in mind there.

Physical examination then is not interesting except for the glands. I think slightly irregular pupils in a man of seventy-seven are of no significance.

The red blood cell counts on the sixth and fourteenth days were 2,550,000 and 2,280,000, that is rather strange in the absence of a story of hemorrhage. I think that the first count of four million probably was an error.

The blood is interesting in comparison with the next episode. The color index according to the Tallqvist scale is high and the red counts are lower than one would expect in view of the history. There is no description of the smear. The differential count shows a slight but absolute lymphocytosis, and there are some immature cells interpreted as lymphocytes. Taken in connection with the glandular enlargement that blood smear would suggest a lymphoid tumor of a cellular type and certainly I think would justify a diagnosis of aleukemic leukemia, taken also in connection with the severe anemia. The blood picture is not that of a Hodgkin's disease, where, having gone this length of time, one would expect to find the lymphocytes reduced instead of increased.

The skin lesion may of course have been due to lymphoid involvement of the skin but there is no more mention of it and we might as well leave it as it is.

The history and physical examination on the second admission are essentially the same as previously except for sore tongue and the weakness and so on, presumably due to anemia of some sort. The scanty hair I think one can dismiss because he was known to have had considerable radiation in these regions.

We again have a very severe anemia which shows this time a relative lymphocytosis, but there is no actual increase of lymphocytes. There are less immature cells this time, not classified. The picture might be consistent with the anemia of aleukemic leukemia. In this condition we frequently get an anemia which shows marked variation in the size of the red cells, sometimes a true macrocytic hyperchromic anemia, one very difficult to distinguish from pernicious anemia. The test that has been used frequently is a therapeutic one, with the use of liver. It is rare in this type of anemia associated with leukemia to get variation in the shape of the red cells. On the other hand this patient had a sore tongue, suggesting vitamin B deficiency. It impresses me when I go around and look at patients with long-continued chronic disease, especially when I look at their tongues, that there frequently is evidence of vitamin B deficiency with or without anemia. I refer especially to patients with diseases such as ulcerative colitis or long-drawn-out gastrointestinal malignancy or patients with difficulty in swallowing or with long chronic illnesses. These

patients would all be benefited by vitamin B by mouth. This patient, then, possibly suggested pernicious anemia and he was treated with highly purified liver extract which produced no apparent results. This was evidently not a potent material because two weeks after admission treatment with liver that was potent was followed by excellent response. There was marked improvement in strength and appetite, almost complete disappearance of the soreness of the tongue, the red count rose to two million with twenty-six per cent reticulocytes, that is about two hundred and sixty thousand reticulocytes, which is a maximal response. We see later that his red count rose to four million. In other words, he had an anemia which gave the specific response to liver as shown by maximal reticulocyte response for the red cell level and the problem is simply whether he had two diseases or whether this pernicious-like anemia was secondary to his underlying disease. It is rather ridiculous for one to try to decide because there is, I think, no solution of the problem. I doubt if Dr. Mallory can tell us. However, the description of the patient does not suggest a typical idiopathic pernicious anemia.

Could radiation so affect gastric activity as to produce pernicious anemia? I do not think I have ever seen it as a result of radiation alone. I wonder what Dr. Holmes will say. It may be that his diet had been grossly wrong but there is nothing in the history to suggest that. Finally he may have infiltration of the stomach with tumor tissue, with diminishing gastric activity with resulting pernicious anemia due to the lack of an intrinsic factor. It seems futile to speculate about this.

When we have a patient with marked headache and a probable leukemia or gland tumor such as this it is wise to think of some central nervous system involvement and it may even include a real leukemic meningitis. I got caught that way once, not paying enough attention to the headache. I will speak of that in a moment.

The problem in this last admission is why he had headaches and blurring vision and what actually caused death. The blurring of vision may be due to retinal hemorrhages as a result of anemia. There is no note of hemorrhages elsewhere. The hemorrhage is noted as subconjunctival and therefore I think it is fair to assume that it was due to increased intracranial pressure. I am going to guess that he had not a discrete involvement in the brain but a diffuse involvement around the meningeal vessels with this tumor, and that he had what may really be called a leukemic meningitis. There is no detailed account of the death, and it is not important in this case. I assume we can take it that he did have a terminal pneumonia in addition to everything else. I should say, then, that he had clinically a leukemia which pathologically might be described as lymphoblastoma of some type, that he had an involvement of the meningeal vessels amounting to a leukemia meningitis, and that he had a symptomatic or secondary pernicious anemia.

Clinical Discussion

Dr. Bernard M. Jacobson: I took care of this patient during his second admission while he was in Ward 4 and we had all the data about his having been discharged on a previous admission with a biopsy diagnosis of lymphosarcoma. While he was attending the Tumor Clinic for continued x-ray treatment Dr. Chapman noticed the appearance of other symptoms, weakness, dyspnea, sore tongue and the onset of paresthesia in the hands. Dr. Chapman, knowing that I was interested in pernicious anemia, got me to see him at the time of his second admission. The physical examination, the history and the blood findings could not be distinguished from that of idiopathic pernicious anemia. It is interesting in retrospect that he did have at least two high color indexes at the time of previous admission but not nearly so high at the time of the second admission.

After his discharge from Ward 4 I followed him in part during the next four months. I did not give him any parenteral liver treatment because he seemed to do very well on whole liver and domestic liver by mouth. About two months after discharge at a time when the red blood cell count was well over four million he came in with a story that was typical of acute gout involving one metatarsal phalangeal joint. He had never had any arthritic symptoms in his life before and no family history of gout. A few weeks later he came in with another story of an attack of acute metatarsal phalangeal inflammation. I saw him after the second attack when the joint was subsiding. He had very slight edema and slight brownish discoloration of the skin. I did not modify the diet in any way and sent him to the house for the third admission.

A Physician: I should like to ask one question. Must we make a diagnosis of idiopathic pernicious anemia on the basis of a maximal reticulocyte response to treatment? I have been asked that a number of times and I am not sure whether the therapeutic test is a final test of idiopathic pernicious anemia. I should like to hear a comment on that.

Dr. Francis T. Hunter: I should like to ask Dr. Jacobson whether the cell volume was high.

Dr. Jacobson: The volume index was as high as you will ever find it, around 1.6. It is far higher than we see it in the myeloid leukemias or lymphoblastomas, which rarely run over

1.3. I think it is the experience of most people that the effect of liver extract in large doses in the non-pernicious anemia, macrocytic anemias—if any effect at all is noticed—is a very irregular one, and I doubt whether anybody has observed a true reticulocyte crisis with a specific gain of red cells and improvement of symptoms in any disease other than something like pernicious anemia. I have one functional test regarding this diagnosis that I will mention later.

DR. ARLIE V. BOCK: Dr. Richardson's diagnosis of leukemia seems to me to be almost impossible in view of the response to liver and the great regeneration of red cells. We look upon leukemia as a widespread bone marrow tumor involvement with destruction of normal blood forming machinery and if it is a leukemia I should think it would be most unusual to get this response. I think he has two independent diseases.

CLINICAL DIAGNOSES

Lymphosarcoma.
Pernicious anemia?

DR. WYMAN RICHARDSON'S DIAGNOSES

Aleukemic leukemia (lymphoblastoma).
Leukemic meningitis.
Symptomatic (secondary) pernicious anemia.

ANATOMIC DIAGNOSES

Lymphoblastoma, ? giant follicular type, with involvement of the spleen, kidney, bone marrow and lymph glands.
Bronchopneumonia.
Septicemia, staphylococcus aureus.
Hydrothorax, bilateral.
Pulmonary edema.
Pulmonary atelectasis.
Pleuritis, chronic fibrous, left.
Arteriosclerosis; marked coronary; moderate aortic.
(Gout.)
Prostatic hyperplasia.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: There is one prognosis which Dr. Richardson made which is absolutely correct and the safest he could have made; that is that the pathologist would not be able to make a diagnosis. The one thing that most of us expected to find in this patient was a marrow full of cells whatever they might be; if he had pernicious anemia, megaloblasts; if leukemia, lymphoblasts or some form of lymphocyte. The rather surprising result of the autopsy was a completely aplastic bone marrow. I think you can see pretty readily with this low power that the fat cells have been almost entirely replaced but that the actual marrow cells are very few and far between. What makes up the great majority of the marrow is a sort of gelatinous, edematous fibrous tissue, only here and there can you see a few small collections of marrow cells. I suppose the number of marrow cells is reduced to one-fiftieth or one-hundredth of the normal amount. In other words a very severe grade of aplasia or atrophy of the bone marrow is present.

The diagnosis of lymphoma had been made a few years ago and at that time it was considered to be of the sarcomatous type. On reëxamination it is evident that the original diagnosis as to type is wrong; it should have been called giant follicular lymphoma rather than lymphosarcoma. That is a diagnosis with which many of you are probably still relatively unfamiliar. It is only within the last five years that we have been recognizing them. I have no doubt that, if we were to go back over our entire lymphoma material, we would pick up a great many cases that have been classed under other diagnoses.

Follicular lymphomas may logically be considered so nearly the reproduction of normal lymphoid tissue that true, albeit somewhat typical, follicles are found. For that reason it is easily missed and may be passed up as simple hyperplasia. In other instances the follicles are so numerous, so large, and so closely packed together that unless the specimen is carefully examined with a very low power their presence will not be recognized.

The general principle that well differentiated tumors are of low malignancy in comparison with poorly differentiated ones seems to hold for these neoplasms. Ewing goes so far as to regard some of them as benign, our own experience is that they always recur though the interval, as in this case, is apt to be a long one. Five to ten years of good health between primary tumor and death is by no means uncommon. In the recurrences the growth is not so typical and the differentiation is much poorer. That was the case here. Lymph nodes, spleen and kidney were extensively involved but only the kidney showed characteristic follicles.

The puzzle of the case lies in the bone marrow. Did the patient at one time have leukemic infiltration of his marrow so extensive as to displace all the normal cells? I feel that is a possibility since I have seen another case of leukemia—proved by bone marrow biopsy—come to autopsy with a totally aplastic marrow. Did he ever have pernicious anemia? The autopsy gives us no clue. Did he develop a primary aplastic anemia or a secondary one due to excessive x-ray or to Fowler's solution? Those possibilities certainly cannot be ruled out. My personal preference, however, lies with the hypothesis of leukemia. I should like to hear Dr. Holmes' opinion. Could x-ray treatment produce this result in a normal marrow or in pernicious anemia, or do we have to assume that there was a lymphomatous involvement of the bone marrow first and the x-ray destroyed that

and left nothing behind? How do you feel about it, Dr. Holmes?

DR. GEORGE W. HOLMES: I cannot definitely answer the question. Of course we do get anemias as an end result in these cases of leukemia that have been treated for a long time. Then there are cases of polycythemia which respond very readily to small doses of x-ray showing that irradiation does affect the bone marrow. The question may well be raised but it cannot be answered at present.

Dr. Richardson raised a question that is worth commenting upon, the question of a change in the stomach causing the anemia. We should have had an x-ray examination of the stomach. We are inclined to neglect these studies in patients who have been with us for a long time.

DR. MALLORY: The stomach was entirely negative at autopsy. It was a perfectly logical thing to think of, of course.

A PHYSICIAN: Was the head examined?

DR. MALLORY: No.

DR. HOLMES: I should like also to comment on the reaction following the treatment to the head. It is quite common in radiation over the brain to have such a reaction and it is possible that the treatment did increase the intracranial pressure but in this case the situation was desperate and something had to be done.

DR. MALLORY: Dr. Weiss has recently been collecting cases of leukemia, and finds that central nervous system involvement is more common than not, strikingly more frequent than is generally supposed.

A PHYSICIAN: Were there many specimens of bone marrow examined, Dr. Mallory?

DR. MALLORY: The sternum and vertebrae.

A PHYSICIAN: I wonder how he could have such a generalized aplastic anemia and maintain such a respectable blood picture.

DR. MALLORY: He was in five weeks and the last red count was taken a number of weeks before his death.

A PHYSICIAN: Did he maintain as high a blood count as that at the end?

DR. MALLORY: I cannot answer that.

A PHYSICIAN: Did they make a diagnosis of leukemia?

DR. MALLORY: No.

A PHYSICIAN: Do you think he has lymphosarcoma?

DR. MALLORY: I am inclined to think he had leukemia, but there is no positive evidence of it from the autopsy. I am inclined to think he had true pernicious anemia. What do you think, Dr. Jacobson?

DR. JACOBSON: At the end of the second admission the red blood cell count had gone up to three million and the glands began to recur. On Dr. Hunter's advice we gave him more treatment.

I should like to ask Dr. Mallory if he can distinguish the stomach in pernicious anemia from a normal one?

DR. MALLORY: The autopsy was done ten hours postmortem and it would be quite impossible.

DR. JACOBSON: We have one method that we have developed in this hospital for the estimation of the hematopoietic potency of various materials and various organs. We have found in one individual, a person dying of some other diseases with no anemia during life, that his liver contained 150,000 guinea pig units of activity per 100 grams of liver. We have assayed the livers of two patients dying of pernicious anemia in relapse and in both these instances there were less than 500 units of activity per 100 grams of liver. In this particular patient who died in what we might call partial remission, the content was 47,000 and these figures all agree with the results of human assays of such livers in three other investigations; namely, that the normal liver is about as good as hog or beef liver; the pernicious anemia in relapse-liver is practically devoid of activity and the pernicious anemia in remission-liver is active but not nearly so active as the normal human liver. So we have some functional evidence that he had pernicious anemia.

CASE 21282

PRESENTATION OF CASE

A fifty-two year old Irish laborer entered complaining of abdominal pain of three days' duration. (An accurate history could not be obtained because of the condition of the patient.)

Three days before entry, early in the afternoon, the patient developed generalized lower abdominal pain. This pain was his chief symptom and was severe enough to keep him awake at night. He had lost his appetite but had not vomited. His bowels had not moved during the past two days. He had been followed by a physician who had given him an enema the day before entry. There was no previous history of abdominal pain. He denied ever having been sick before.

Physical examination showed a very acutely ill man with a very foul breath. His tongue was dry. The heart and lungs were negative. The blood pressure was 100/60. His abdomen was very stiff and uniformly tender, not more on one side than the other. Rectal examination was negative. The reflexes were normal.

The temperature was 101.6°, the pulse 140. The respirations were 34.

No urine could be obtained on admission. The blood showed a white cell count of 34,000.

An abdominal x-ray film showed no free air beneath the diaphragms. No fluid levels were seen in the unusual gas shadows of the abdomen.

About 4 liters of 5 per cent glucose was administered intravenously. Another attempt to

procure a urine specimen by catheterization three hours after the intravenous was unsuccessful. Only a few drops of mucus but no urine was obtained. He rapidly went downhill, became comatose and died approximately twelve hours after admission without having regained consciousness.

Differential Diagnosis

Dr. Leland S. McKittrick: The positive findings as I see them are severe generalized pain of three days' duration, and no nausea or vomiting in a man who had never been sick before, and whose bowels had not moved for two days. In addition to that we have an acutely ill patient with distress, dry tongue, blood pressure 100/60, which I assume to be the result of his collapse rather than a normal blood pressure, and who has the physical signs of peritonitis. He has passed no urine since his admission to the hospital.

The inability to get urine, and I presume we can assume that the catheter went into the bladder all right, might be due to one of three things: obstruction at the base of the bladder, so that no urine could get into the bladder, frank rupture of the bladder, or failure of the kidneys to excrete it. It is perfectly possible that it might be due to the fact that the kidneys were not excreting urine during the period between the last voiding and the time he was catheterized. I do not think we have anything here to make us assume that he had an obstructing lesion shutting off his ureters. One might say that he had a ruptured bladder with resulting peritonitis. I believe that if you catheterized such a bladder and there was no urine in it there would be blood in what was obtained. Therefore, I believe the inability to get urine was probably due to the fact that urine was not being excreted during the particular time between his last voiding and catheterization. If that is the case it might possibly be secondary to the terminal stages of an abdominal condition. There again without any more lead than we have in the history or physical examination one cannot do much more than speculate as to the cause of peritonitis which I also assume is present.

One may have a peritonitis of the chemical type such as occurs in acute pancreatitis from the bile and blood-tinged fluid, or it might have an infectious origin. I do not believe there is any reason for assuming it to be a chemical peritonitis. I do not believe he has acute pancreatitis because he is not distended, is not vomiting, and has not the tenderness along the pancreas that usually occurs with pancreatitis. I do not believe it is blood in the peritoneal cavity because I cannot quite fit together an intelligent story which would give such a picture. If it is a septic peritonitis one might get it either from perforation of an organ or from gangrene.

This is a rather unusual story for appendicitis with perforation and death. It is possible of course. It is the most common cause but where your cases are selected you cannot very well go on the law of chances. A ruptured diverticulum of the sigmoid or a perforated peptic ulcer might give the same picture. I am interested in the unusual gas shadows.

Dr. George W. Holmes: There is not much to see in this film. It was taken probably with the patient lying on the back and might miss free air in the abdominal cavity. He evidently had a tube in the stomach. The shadow that you see on the left side is probably gas in some part of the gastrointestinal tract. We would not think it was particularly unusual. They do not say what was obtained with the stomach tube.

Dr. Henry H. Faxon: Nothing of any great significance.

Dr. Holmes: They do not say what results the physician got from the enema.

Dr. Faxon: The bowels had not moved for two days.

Dr. McKittrick: That does not help very much. The onset certainly is not typical of a perforated ulcer.

Dr. Faxon: As I remember the story the present illness was more than three days. He had discomfort in the lower abdomen five days before and felt that the best thing to do was to take something for it. Accordingly he took a sufficiently substantial amount of liquor to become unconscious of pain and kept himself in that state for three days. When he became conscious he found the pain still there and again "loaded up" and carried on until the day of entry, when he was no longer strong enough to go out for any more, so his friends brought him a half pint of straight whiskey. That was one reason we could not get a better story. I think he had more pain than he complained of.

Dr. Robert G. Glendy: Upon further investigation of the case after death I learned from the doctor who took care of him outside and who saw him three days before he came in here, that the pain was chiefly in the upper abdomen and that he had considerable precordial pain with some nausea and vomiting.

Dr. McKittrick: I think we had better stop. It is getting more complicated. I still do not think that he had perforated ulcer. I cannot exclude it. I cannot exclude anything. I can only rationalize a little bit and be wrong. The other thing that presents itself to me is to try to coördinate the unusual complete anuria with the abdominal findings. With a pretty good cardiovascular system and good kidneys, this man could not have been as well so he said, never having been sick before, and have a com-

plete shutdown of the kidneys, without some very dramatic accident. He had a 34,000 white count. He is tremendously dehydrated and it does not mean much but I do think that one of the causes of a very short terminal illness, with obscure general abdominal pain and widespread evidence of peritonitis, is peritonitis secondary to gangrene of the bowel. A mesenteric thrombosis to me would fit the whole picture of anuria and an early death after a relatively short and obviously very serious illness. I can only say that I believe the man has no mechanical obstruction to his ureters. I believe he died with a peritonitis. I believe his anuria is secondary to the peritonitis plus another fundamental process and my first choice as to the cause of his peritonitis would be a mesenteric thrombosis.

DR. FAXON: When he came in we felt he had a peritonitis. I think that we laid less stress on the failure to get a specimen of urine by catheter than has been brought out in the present discussion. I could not explain the picture, but feeling that the man had peritonitis, with the most likely etiology on the law of chances a ruptured appendicitis — although it was certainly atypical for that—we brought him to the operating amphitheater. His condition at that time was so critical it was obvious that operative interference would certainly end fatally, so we felt that it was wiser to carry him along with intravenous therapy and see if he would respond. Instead of responding he went steadily and rapidly downhill without operation.

CLINICAL DIAGNOSIS

Peritonitis, ? cause.

DR. LELAND S. MCKITTRICK'S DIAGNOSIS

Mesenteric thrombosis with secondary peritonitis and anemia.

ANATOMIC DIAGNOSES

Aneurysm of the aorta, dissecting.
Hemorrhage into myocardium about right coronary artery.
Central necrosis of liver.
Cloudy swelling of the kidneys.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: Obviously the data are entirely inadequate for a diagnosis on this case. At Dr. Faxon's suggestion we put in the only lead that there was and emphasized it a little bit. If we had had some of the information which Dr. Glendy has since picked up I think we could have given you a better story. What he had was a dissecting aneurysm of the aorta running all the way from the aortic ring down to the iliacs. Dissection did not extend down any of the branches of the abdominal aorta but I think it is fair to assume that they were probably functionally occluded by pressure at their mouths. The kidneys were not very remarkable grossly. Microscopically they show a diffuse cloudy swelling of the parenchyma. The most definite change and one which I think is strong evidence for circulatory insufficiency was found in the liver where the central two thirds of every lobule was completely necrotic. We have seen this lesion with particular frequency in cases of acute bleeding duodenal ulcer, so that I feel that we have good histologic evidence of insufficient circulation of the liver and suggestive though not certain histologic evidence of interference of circulation of the kidneys. I believe that is the only way in which the symptoms can be connected up. There was little or no blood circulating through the kidneys and consequently no urine was put out. The bladder and prostate were entirely negative.

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LOVING DARKNESS RATHER THAN LIGHT

THE resolution asking for the appointment of a commission to study the condition of medical education in Massachusetts, which has been before a committee of the legislature for several weeks, has, we understand, not met the approval of influential members of the Legislature. A full setting forth of the basis for this action has not been made public but a discussion of the matters involved is pertinent at this time.

Substantially the commission was to be a fact finding body. Possible objection might be raised on the grounds that the commission was incompetent, or unnecessary or expensive. The actual objections were on the grounds of expense, prejudice on the part of the commission and lack of need for the investigation as expressed at the hearing. The question of expense need not be considered: if the investigation were worth anything it would be worth the fifteen hundred dollars for clerical services, as the commission was to be unpaid.

The question of competence does not need lengthy discussion. The commission was to consist of fifteen members: one senator, three representatives; two physicians appointed by the Governor; the Commissioner of Education; the Commissioner of Public Health; the Chairman of the Board of Registration in Medicine; and the deans of the six schools in Massachusetts from which graduates are accepted for examination for the practice of medicine. Until the persons were named and had served, no final judgment could be passed, but the proposed list represents grounds for a reasonable expectation that the report would be intelligent and intelligible.

The chief prejudice was shown by the opponents of the resolve who claimed that the proponents were "tyrants" and that the commission was "packed" against the "small schools", and in the nature of things could not render a fair report. This general insult was gratuitous and unwarranted.

The necessity for the investigation remains to be considered. Are investigations of educational institutions necessary? When? How? By whom? Why? Certainly investigations are necessary and the reports of college and university presidents indicate the self-critical spirit, sometimes a little severe, in which many of the activities of the institutions are reviewed annually. Sometimes a spirit of complacency and self-congratulation is evident, but the possibility for self-criticism exists, and although these documents do not usually receive wide circulation except among alumni, no effort is made to conceal them.

Occasionally there is investigation of another sort, as recently of the University of Chicago by the Illinois legislature. On report of alleged improper activities, it was made promptly and no improper activities were found. Another type of investigation has been made by organizations interested in medical education, because, and it is a matter of record, medical schools were purely commercial organizations in many cases. Commercialism in professional education is intolerable, and the force of public opinion, following the light thrown by these investigations, has caused nearly all of the commercial medical schools to disappear.

It is recognized in scientific circles that a determination of facts should precede action and the beneficial and stimulating effects of knowing the facts can hardly be overestimated. So in recent months there has been undertaken again a careful study of medical education in the United States, and the report is expected within the next year. The report will not include schools of medicine or osteopathy which have refused to be surveyed.

Why should a school refuse to be surveyed? Why should it refuse to make known how it car-

ries on medical education, how it provides instruction for its students, how it upholds the ideals of the medical profession?

The recent attempt in Massachusetts to discredit education suggests, perhaps, that these schools which refuse and apparently fear a survey, have such high standards that their preëminence would subject them to jealous attack. On second thought, however, it seems more probable that they fear exposure because they fear the result of the action of public opinion against matters discreditable.

If their records are clear, they ought to be the first to demand a survey so that all men may know that they have been unjustifiably suspected of selling shoddy medical education, or of sharp practice or of something worse.

PASTEURIZATION OF CERTIFIED MILK OFFICIALLY ACCEPTED

THE American Association of Medical Milk Commissions at its annual meeting in Atlantic City last month recorded two notable achievements; it elected Dr. Milton J. Rosenau as president, and it unanimously accepted the pasteurization of certified milk as an official, although not an obligatory, procedure.

Dr. Rosenau, both as professor of hygiene at the Harvard Medical School and as an accepted leader in public health movements, has long been recognized as one of the uncompromising exponents of safe milk and clean milk. His election, to succeed Dr. Hugh L. Dwyer, is a tribute to his leadership and a guarantee that the Association of Milk Commissions will continue to be, as it has been in the past, a guiding spirit in the progressive improvement of the standards of milk production.

The fact that improvement in the production of so much of our milk supply has tended to lessen the vital importance of certified milk in certain of the uses of milk, such as infant feeding, must not cause us to lose sight of the fact that certified milk has for over forty years set a goal and served as a model in this improvement. Moreover, most of the more recent continued advances, except for pasteurization, have been inaugurated by certified milk.

The certification of milk was created at a time when raw milk was considered as an asset, particularly in the feeding of infants, and the raw state was consequently considered as a sine qua non in its production. It may be that pride in the ability conscientiously to combine safety with rawness was one of the factors which delayed for so long the acceptance of pasteurization; in any event, for many years, according to the standards of the Association, certified milk could not be pasteurized milk.

In the face of this prejudicial fact it is a matter of interest and of considerable local pride that the Boston Medical Milk Commission, some four or five years ago, not only allowed but encouraged its producers to market certified pasteurized milk in defiance of the regulations of the Association, and put the stamp of its approval on the product. Some efforts were made to discipline our Commission for its revolutionary attitude, but strong in its convictions it continued its independent course, and the matter was allowed to drop.

Acceptance of pasteurization was the wisest course for the Association to follow. Certified milk has a distinct place in the milk industry, and meets a distinct demand from the consumer for a milk produced as nearly ideally as possible; but without pasteurization, in this day when pasteurization is a household by-word, it suffered an insuperable self-imposed handicap, except in such sections as our own where the tradition had been broken. With this handicap removed certified milk may continue to set the standard by which all good milk is produced.

The only unhappy note at the meeting this year was introduced by the resignation of Dr. Harris Moak, for a number of years the secretary and treasurer of the Association.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

PHELPS, O. DRAPER. A.B., M.D. Dartmouth Medical School 1907. F.A.C.S. Urologist, Worcester City Hospital. Cystoscopist, Memorial Hospital, Worcester. Assistant Surgeon, Holden District Hospital. Urologist, Belmont and Louis Pasteur Hospitals. Consultant in Urology, St. Vincent Hospital, Worcester. His subject is "Hemorrhagic Cystitis and Tuberculosis of the Prostate." Page 43. Address: 27 Elm Street, Worcester, Massachusetts.

LELAND, HAROLD L. M.D. Boston University School of Medicine 1917. F.A.C.S. Assistant Professor of Genito-Urinary Surgery, Boston University School of Medicine. Surgeon, Massachusetts Memorial Hospitals. Urologist, Lowell General and St. Joseph's Hospitals. Urologist, Lowell Cancer Clinic. Chief, Lowell Genito-Urinary Clinic. His subject is "Vesicointestinal Fistula." Page 44. Address: 226 Central Street, Lowell, Massachusetts.

BALDRIDGE, ROBERT R. A.B., M.D. Harvard University Medical School 1925. Junior Assistant Visiting Surgeon and Surgeon to Out Patient Department, Rhode Island Hospital. Assistant Surgeon in Urology, Chapin Hospital, Providence. Surgeon to Rhode Island State Hospital. His subject is "A Case of Congenital

Hypertrophy of the Verumontanum." Page 46. Address: 454 Angell Street, Providence, Rhode Island.

MERRILL, EARL S. B.A., M.D. Harvard University Medical School 1920. F.A.C.S. Chief Surgeon, Urological Service, Eastern Maine General Hospital. His subject is "Hydronephrosis—Report of a Case." Page 49. Address: 15 Forest Avenue, Bangor, Maine.

PEIRSON, EDWARD L., JR. B.S., M.D. Harvard University Medical School 1925. F.A.C.S. Urologist, North Shore Babies' Hospital, Salem, and Lynn Hospital, Lynn. Assistant Urologist, Salem Hospital. His subject is "Transurethral Resection of the Internal Sphincter in a Certain Type of Cord Bladder." Page 50. Address: 33 Summer Street, Salem, Massachusetts.

CHUTE, RICHARD. A.B., M.D. Harvard University Medical School 1927. F.A.C.S. Assistant Urologist, Massachusetts General Hospital. His subject is "The Recurrence of Benign Obstructing Prostates Years After Prostatectomy." Page 55. Address: 352 Marlborough Street, Boston, Massachusetts.

JENKINS, RALPH H. M.D. Medical College of Virginia 1916. F.A.C.S. Assistant Clinical Professor of Urology, Yale University School of Medicine. Attending Urologist, New Haven Hospital. Address: New Haven Hospital, New Haven, Connecticut. Associated with him is

DEMING, CLYDE L. B.A., M.D. Yale University School of Medicine 1915. F.A.C.S. Clinical Professor of Urology, Yale University School of Medicine. Their subject is "Cysts of the Testicle." Page 57. Address: New Haven Hospital, New Haven, Connecticut.

BAUMGARTNER, LEONA. A.B., M.A., Ph.D., M.D. Yale University School of Medicine 1934. Instructor in Bacteriology 1926-1928, University of Montana. Research Fellow, Kaiser Wilhelm Institute for Psychiatry, Munich, Germany, 1928-1929. Assistant in Pediatrics, Cornell Medical College 1934-. Her subject is "Edwin Klebs. A Centennial Note." Page 60. Address: New York Hospital, 525 E. 68th Street, New York City.

BOWMAN, KARL M. A.B., M.D. University of California 1913. Assistant Physician, Bloomingdale Hospital 1915-1921. Chief Medical Officer, Boston Psychopathic Hospital 1921-. Assistant Professor of Clinical Psychiatry, Boston University School of Medicine. Special Instructor in Social Psychiatry, Simmons College School of Social Work. His subject is "Progress in Psychiatry for 1934." Page 63. Address: 74 Fenwood Road, Boston, Massachusetts.

MASSACHUSETTS LEGISLATIVE NOTE

House 2245

Resolve providing for an investigation and study by the Commissioner of Public Health of the laws relative to public health and to the establishment and administration of a system of health insurance.

Report, ought to pass, filed in House, July 3, 1935.

Ordered to third reading in House and passed to be engrossed, July 8, 1935.

Referred to next annual session in House July 9, 1935.

MISCELLANY

TUFTS COLLEGE MEDICAL SCHOOL GRADUATES—JUNE 17, 1935

(Address is in Massachusetts unless stated elsewhere)

Abate, Frank Joseph, Jr., Ph.B. (Boston Coll.), 36 Monument Sq., Charlestown.

Abrams, Herbert, B.S. (Univ. of Virginia), 10 Staples St., Lowell.

Aisner, Mark, A.B. (Harvard Univ.), 36 Fessenden St., Boston.

Amrhein, Leo Francis, Ph.B. (Boston Coll.), 55 Brunswick St., Roxbury.

Asali, Louis Anthony, 12 Chatham St., Portland, Me.

Bagnulo, Edmund Gillis, Ph.B. (Boston Coll.), 44 Vane St., Revere.

Baldwin, Joseph Michael, B.S. (St. Bonaventure's Coll.), 10 West St., Simsbury, Conn.

Barker, Richard Lewis, A.B. (Bowdoin Coll.), 73 East Broadway, Derry, N. H.

Beck, Sidney, A.B. (Harvard Univ.), 20 Eutaw St., East Boston.

Binder, Harold Jacob, A.B. (Harvard Univ.), 188 Walnut Ave., Roxbury.

Bixby, Harriet, B.S. (Tufts Coll.), 70 Pleasant St., North Andover.

Bloom, David Irving, B.S. (Conn. State Coll.), 43 Westland St., Hartford, Conn.

BoDine, Charles Elliott, A.B. (Univ. of Oregon), 2439 North East 16th Ave., Portland, Ore.

Bowles, George Ellsworth, B.S. (Tufts Coll.), Plymouth, N. H.

Boyd, Kenneth Taylor, A.B. (Univ. of Michigan), 38 Greenwood St., Amesbury.

Brown, Alice Maude Ruth, B.S. (Tufts Coll.), Welsford, N. B., Canada.

Buccheri, Francis Salvatore, B.S. (Tufts Coll.), 105½ Jackson St., Lawrence.

Buckhout, George Atherton, Pine St., South Hadley.

Burke, Edward William, B.S. (Boston Coll.), 17 Rose Garden Cir., Brighton.

Campbell, Clayton C., Jr., B.S. (Univ. of Oregon), 387 Wisconsin Ave., Long Beach, Calif.

Carey, Edmund Lawrence, A.B. (Boston Coll.), 59 South St., Quincy.

Carolan, James Francis, A.B. (Boston Coll.), 117 Regent St., Roxbury.

Carr, Francis Henry, A.B. (Harvard Univ.), 57 East Border Rd., Malden.

Carr, William Michael, Ph.B. (Boston Coll.), 10 Thayer St., Hingham.

Centrone, Patrick Anthony, B.S. (Univ. of Pittsburgh), 253 West Carlisle St., New Haven, Conn.

Chamberlin, Eugene Francis, B.S. (St. Bonaventure's Coll.), 279 Northern Blvd., Albany, N. Y.

Cohen, William Bernard, B.S. (Mass. State Coll.), 558 Chestnut St., Springfield.

Cotter, Joseph Robert, A.B. (Boston Coll.), 3 Wallace Ct., Charlestown.

Crane, George Edward, A.B. (Brown Univ.), 129 Nelson St., Providence, R. I.

Daniels, Samuel David, B.S. (Tufts Coll.), Sweden St., Caribou, Me.

Davis, Paul Vincent, B.S. (Georgetown Univ.), 25 Clark St., Worcester.

DeMello, Joseph, A.B. (St. John's Coll.), 40 Bliss St., South Dartmouth.

Deutsch, Emmanuel, B.S. (Tufts Coll.), 249 Washington St., Dorchester.

Dias, John Felisberto, Jr., B.S. (Tufts Coll.), 374 Clinton St., New Bedford.

Dretler, William Morris, B.S. (Boston Univ.), 89 Gainsboro St., Boston.

Dunn, Raymond Anthony, B.S. (Tufts Coll.), 23 Brigham Pk., Fitchburg.

Edwards, Jesse Efrem, B.S. (Tufts Coll.), 18 Georgia St., Dorchester.

Faber, David, B.S. (Univ. of New Hampshire), 20 Orange St., Nashua, N. H.

Farren, Edward Bain, B.S. (Harvard Univ.), 55 Fairmont Ave., Brockton.

Feinsilver, Oscar, M.A. (Clark Univ.), 176 Ruthven St., Roxbury.

Feldman, Noah Norman, A.B. (Univ. of Michigan), 28 Ridgewood Ave., Holyoke.

Feldman, Theodore, B.S. (Tufts Coll.), 52 Deering Rd., Mattapan.

Fiske, Reginald Earle, 505 North River Rd., Manchester, N. H.

Ford, Francis Weldon, B.S. (Univ. of Notre Dame), Casselton, North Dakota.

Fraser, Clarence Kendall, Ph.B. (Holy Cross Coll.), 50 St. James Ave., Somerville.

Fratantuono, Frank Domenic, B.S. (Tufts Coll.), 21 Hope St., North Providence, R. I.

Freedman, Herman Sumner, A.B. (Harvard Univ.), 106 Naples Rd., Brookline.

Gallivan, John Norman, A.B. (Tufts Coll.), 230 North Wall St., Meriden, Conn.

Gariepy, Alonzo Osias, B.S. (Tufts Coll.), Summer St., Barre.

Girouard, Fernand Louis, B.S. (Conn. State Coll.), 280 Pleasant St., Willimantic, Conn.

Gorman, Richard Joseph, A.B. (Boston Coll.), 182 Main St., Watertown, Mass.

Gorney, Arthur Joseph, A.B. (Harvard Univ.), 37 Mayflower Rd., Newton.

Gould, William Christopher, Jr., 63 Russell St., Worcester.

Green, William Richard, A.B. (Boston Coll.), 121 Temple St., West Roxbury.

Guy, John Timothy, B.S. (Univ. of New Hampshire), 11 Pine St., Somersworth, N. H.

Hagopian, Peter Bedros, B.S. (Tufts Coll.), 50 Oak St., Lawrence.

Hall, Leonard John, 54 Seventh St., Lowell.

Harrington, Edward Carey, B.S. (New York Univ.), 200 Pine st., Holyoke.

Hart, Virgil Cooley, A.B. (Oberlin Coll.), Windham St., Garrettsville, Ohio.

Haskell, Albert Tuttle, 127 Park St., Livermore Falls, Me.

Hayes, Paul Thomas, B.S. (Bowdoin Coll.) 6 Summer St., Ipswich.

Healy, John Francis, A.B. (Boston Coll.), 14 Tuttle St., Boston.

Hecker, Harry, B.S. (Tufts Coll.), 415 Broad St., Valley Falls, R. I.

Hennessy, Kathryn Anne, 31 Hamilton Ave., Auburn, N. Y.

Hoey, Patrick Henry, B.S. (Harvard Univ.), 30 Montcalm St., Oswego, N. Y.

Hood, Wilfred Thompson, Ph.B. (Brown Univ.), 91 Hobart St., Brighton.

Hylan, Nathan Wicker, A.B. (Univ. of Michigan), 391 William St., Stoneham.

Kaschub, Robert William, A.B. (Wesleyan Univ.), 91 Linsley Ave., Meriden, Conn.

Kasparian, Napoleon Der, B.S. (Tufts Coll.), 310 Talbot Ave., Dorchester.

Kelleher, John Joseph, Jr., B.S. (Tufts Coll.), 61 Eutaw St., Lawrence.

Kennan, Fred John, B.S. (Tufts Coll.), 1 James St., Boston.

Komu, Shizue Helen, A.B. (Univ. of Hawaii), Box 125, Aiea, Oahu, Hawaii.

Koufman, William Bernard, B.S. (Coll. of William and Mary), 60 Clinton Rd., Brookline.

LaBrecque, Frederick Charles, A.B. (Boston Coll.), 42 Avon St., Taunton.

Lamoureux, Eugene Edward, B.S. (Conn. State Coll.), 35 Capen St., Windsor, Conn.

Lamphier, James Andre, A.B. (Harvard Univ.), 195 Lake St., Brighton.

Levin, Orrin, B.S. (Coll. of William and Mary), 27 Elba St., Brookline.

Of the Class of 1934—Levy, Samuel Howard, B.S. (Univ. of Georgia), 1309 Fairfield Ave., Bridgeport, Conn.

Lipton, Joseph Harold, B.S. (Harvard Univ.), 104 Crawford St., Roxbury.

Lord, Charles Rogers, Jr., B.S. (Bates Coll.), 83 High St., Ipswich.

Maher, Joseph Perkins, A.B. (Holy Cross Coll.), 233 Walnut St., Dedham.

Mahoney, Alfred Vincent, A.B. (Boston Coll.), 2 Franklin St., Chelsea.

Mahoney, Frank Aloysius, Jr., A.B. (Boston Coll.), 42 Crescent Ave., Chelsea.

Manley, James Sylvester, Ph.B. (Boston Coll.), 91 Mill St., New Bedford.

McCarthy, Edward Aloysius, B.S. (Tufts Coll.), 75 Salem St., Malden.
Montano, Charles Carl, B.S. (Tufts Coll.), 148 Brown St., Hartford, Conn.
Moore, Donald Bernard, A.B. (Holy Cross Coll.), 271 Humphrey St., New Haven, Conn.
Morton, Edward Augustus, B.S. (McGill Univ.), 35 Bank St., St. Albans, Vt.
Mullins, Stanley St. Clair, Ph.G. (Mass. Coll. of Pharmacy), 32 Pine St., Cambridge.
Murphy, William Francis, Ph.B. (Brown Univ.), 241 Salem St., Malden.
Norton, Louisa Macdonald, A.B. (Radcliffe Coll.), 354 North Underwood St., Fall River.
O'Brien, Francis Robert, A.B. (Yale Univ.), 501 Beacon St., Boston.
Ockert, Raymond George, B.S. (Tufts Coll.), 166 County St., Attleboro.
Omori, Ethel Hideko, Box 127, Mountain View, Hawaii.
O'Toole, Francis Austin, A.B. (Georgetown Univ.), 175 Cedar St., Clinton.
Owen, Charles Kenneth, B.S. (Tufts Coll.), 92 Elm St., Pittsfield.
Paydos, Walter Michael, Ph.B. (Brown Univ.), 163 Church St. Woonsocket, R. I.
Reilly, Walter John, A.B. (Fordham Univ.), 170 Meadow St., Naugatuck, Conn.
Rubin, Abraham Louis, M.A. (Univ. of Maine), 312 French St., Bangor, Me.
Rudy, Harold, B.S. (Tufts Coll.), 27 Summer St., Revere.
Ryan, Francis James, B.S. (Trinity Coll.), 87 Spring St., Hartford, Conn.
Sayers, Daniel O'Connell, B.S. (Conn. State Coll.), 716 Garden St., Hartford, Conn.
Sayers, John Joseph, B.S. (Trinity Coll.), 197 Preston St., Hartford, Conn.
Showstack, Nathaniel, B.S. (Tufts Coll.), 49 Browning Ave., Dorchester.
Smith, Eugene Francis, A.B. (Boston Coll.), 14 Daniels St., Millis.
Smith, John Ellsworth, B.S. (Tufts Coll.), 285 Front St., Weymouth.
Spinner, Samuel, A.B. (Tufts Coll.), 65 Floyd St., Dorchester.
Thomas, Warren Dustin, 163 City Rd., Turners Falls.
Tompkins, John Butler, B.S. (Wesleyan Univ.), 68 Lunda St., Waltham.
Wies, David, A.B. (Harvard Univ.), 259 Mountain Ave., Malden.
Wight, Donald Gordon, B.S. (Bates Coll.), 363 Main St., South Portland, Me.
Wiklund, Folke Walfred, 15 Moultrie St., Dorchester.
Winkler, Malcolm Albert, A.B. (Brown Univ.), 30 Glen Rd., Providence, R. I.
Wolanske, Stephen, A.B. (St. John's Coll.), 441 Pleasant St., Gardner.
Wonson, John Fletcher, 722 Washington St., Gloucester.
Zeff, Sidney Myer, B.S. (Tufts Coll.), 119 Washington St., Dorchester.

PREVIEW OF SCIENTIFIC PROGRAMS

SIXTY-FOURTH ANNUAL MEETING AMERICAN PUBLIC HEALTH ASSOCIATION AND MEETINGS OF RELATED ORGANIZATIONS MILWAUKEE, OCTOBER 7-12, 1935

More than 2000 professional public health workers from every state in the Union, from Canada and from Mexico, will assemble in Milwaukee the week of October 7 at which time the following organizations will be in convention:

American Association of School Physicians
International Association of Dairy and Milk Inspectors
Conference of State Sanitary Engineers
International Society of Medical Officers of Health
Association of Dairy, Food and Drug Officials
Conference of Wisconsin Health Officers.

From scientific programs totaling 400 presentations on subjects timely in the fields of public health, preventive medicine and sanitation, a few of the highlights are selected.

Mental Hygiene has been forging ahead so steadily in interest that an entire session will be devoted to it this year for the first time.

Papers on Economic Consequences of Ill Health, on Health Department Functions—preventive, nursing, medical care, dental care, hospitalization and maternity care—and the Health Department Relationships to the Activities of the Welfare Department and of the Medical Profession will make up a program on the health department and the social security program. The contributors here will be health officers, economists, practicing physicians, welfare directors and officers of government.

The Fifteenth Anniversary of the Association's Committee on Administrative Practice is to be commemorated by a program in which those who have been most intimately connected with its development will review its history and accomplishments, detail the present status of its work, and cast an eye ahead upon its hopes for the future.

Dr. E. L. Bishop, President of the A. P. H. A., and Dr. Walter E. Brown, President-Elect, are always worth listening to, speaking as they do from a wealth of practical experience and personal philosophy.

Professional Education is the subject for discussion at a luncheon meeting on Wednesday. The committee is prepared this year to make some important announcements of policies. Particularly in view of the new personnel that will certainly be drawn into public health work with the passage of the Social Security Bill, the determination of what constitutes adequate preparation for a career in public health is of paramount concern.

Another luncheon session, now traditional at Annual Meetings, is the one devoted to Diphtheria Immunization.

The many allied organizations and special groups that meet with the American Public Health Associa-

tion add interest and variety to the proceedings. Joint sessions are being arranged by various Sections with the American Association of School Physicians, the International Association of Dairy and Milk Inspectors, the Association of Food and Drug Officials, the Conference of State Sanitary Engineers, the American Association of State Registration Executives. The Association of Women in Public Health, Delta Omega, Johns Hopkins, Massachusetts Institute of Technology and Harvard Alumni, and the Conference of State Laboratory Directors will dine, lunch or breakfast and confer.

The ten Section programs reflect present-day attitudes and are thoroughly infiltrated with social and economic significance. The federal health program is given a large share of attention, and its possibilities and implications are thoroughly covered. The following subjects will indicate the breadth and scope of Section activity:

Public Health Significance of Poisonous Substances in Foods
A Planned Milk Control for State and Nation
Standard Methods for the Examination of Shellfish
An Improved Agar for Use in Making Bacterial Counts from Milk
Proposed Changes in Standard Methods for Bacteriological Examination of Milk
Whooping Cough and Measles
Mentality and Morbidity
Pneumonia as a Community Health Problem
Child Health at Different Levels
Growth and Nutrition
Health Training and Instructions in Schools
Scarlet Fever
Animal Diseases Affecting the Public Health
Recent Progress in Biological Products
Customer Demand for Vital Statistics
Sanitation in Rural and Recreational Areas
Air Conditioning and Industrial Health
Occupational Diseases
Present Status of the Vitamin B Complex
Food Advertising under the Food and Drugs Act
Maternal and Neonatal Hygiene
Adequate Health Service for the Child
Commercial Advertising's Challenge to Health Education
Community Organization for Health Education
FERA Problems
Typhoid Carriers
Institutional Outbreaks of Pneumonia.

The preliminary program will be published in full in the September issue of the American Journal of Public Health.

LIST OF GRADUATES AND HOSPITAL INTERNESHIPS OF THE HARVARD MEDICAL SCHOOL, CLASS OF 1935

Name	Hospital	Service	Dates
Appleton, F. M.	Mary Hitchcock, Hanover, N. H.	Rotating	Jan. '36-Jan. '37
Barrow, W.	Mass. General, Boston	Surgical	July '35-Aug. '37
Bates, O. G.	Children's, Boston	Pathological	June '35-June '36
Beckman, W. W.	Mass. General, Boston	Medical	Jan. '36-Aug. '37
Bell, J. F.	Palmer Mem., Boston	Surgical	July '35-Jan. '36
	Peter Bent Brigham, Boston	Surgical	Feb. '36-July '37
Bell, L. M.	Roosevelt, N. Y. C.	Surgical	Jan. '36-Jan. '39
Bienkowski, J.	St. Francis, Hartford, Conn.	Rotating	July '35-July '36
Billo, O. E.	Lenox Hill, N. Y. C.	Medical	Jan. '36-Jan. '38
Bolanowski, K. J.	St. Luke's, New Bedford, Mass.	Rotating	July '35-July '36
Botsford, T. W.	Children's, Boston	Surgical	Oct. '35-July '37
Brenner, C.	Peter Bent Brigham, Boston	Medical	Oct. '35-July '37
Caffee, H. H.	Duke, Durham, N. C.	Surgical	July '35-July '36
Caldwell, C. W., Jr.	Bellevue, N. Y. C., 4th Div.	Surgical	July '35-July '36
Campbell, E. C.	Mary Hitchcock, Hanover, N. H.	Rotating	July '35-July '36
Campbell, J. B.	Children's, Boston	Surgical	Apr. '36-Jan. '38
Carpenter, P.	Memorial, Pawtucket, R. I.	Rotating	Aug. '35-Aug. '36
Clark, S. D.	Hartford Hosp., Conn.	Rotating	July '35-July '37
Clement, D. H.	Boston City, 4th Service	Medical	July '35-Jan. '37
Cogan, J. R.	Univ. of Chicago Hosp., Chicago, Ill.	Medical	Jan. '36—
Cover, W. L.	Strong Mem., Rochester, N. Y.	Medical	July '35-July '36
Cowan, A. W.	Presbyterian, Philadelphia, Pa.	Rotating	July '35-July '37
Croce, E. J.	Worcester City, Mass.	Rotating	May '35-May '37
Cunney, J. V.	Salem Hosp., Mass.	Rotating	July '35-July '36
Curnen, E. C.	Children's, Boston	Bacteriological	June '35-June '36
Dawson, W. E.	Bridgeport Hosp., Conn.	Rotating	July '35-July '36
Donaldson, G. A.	Mass. General, Boston.	Surgical	Apr. '36-May '38
Dutton, R.	Worcester City, Mass.	Rotating	Aug. '35-Aug. '37
Eddy, M. H.	Bellevue, N. Y. C., 4th Div.	Surgical	Jan. '36-Jan. '38
Filmer, G. A.	Denver General, Colo.	Rotating	July '35-Jan. '37

Flannery, W. E.	Cleveland City, O.	Rotating	July '35-July '36
Forster, D. E.	Johns Hopkins, Baltimore, Md.	Medical	July '35-July '36
French, T. A.	Mercy, Pittsburgh, Pa.	Rotating	July '35-July '36
Friend, D. G.	Peter Bent Brigham, Boston	Medical	Oct. '35-Feb. '37
Frothingham, J. G.	Bellevue, N. Y. C., 1st Div.	Medical	Jan. '36-Jan. '38
Getting, V. A.	Worcester City, Mass.	Rotating	Nov. '35-Nov. '37
Giddings, P. D.	Children's, Boston	Surgical	July '35-Apr. '37
Giffin, L. A.	Bellevue, N. Y. C., 2nd Div.	Medical	July '35-July '37
Greenleaf, H. McC.	Charles T. Miller, St. Paul, Minn.	Rotating	July '35-July '36
Grindlay, J. H.	Mary Hitchcock, Hanover, N. H.	Rotating	July '35-Jan. '37
Gundersen, T. E.	Presbyterian, N. Y. C.	Medical	Oct. '35-Nov. '37
Hadler, A. J.	Peter Bent Brigham, Boston	Medical	Feb. '36-Nov. '37
Hall, D. T.	Peter Bent Brigham, Boston	Surgical	June '35-Oct. '36
Hanford, R. B.	Bellevue, N. Y. C., 1st Div.	Surgical	Jan. '36-Jan. '38
Hawkins, J. W.	Cincinnati General, O.	Rotating	July '35-July '36
Henderson, J. W., Jr.	Beverly Hosp., Mass.	Rotating	Aug. '35-Aug. '36
Hindman, D. H.	Beth Israel, Boston	Surgical	Feb. '36-Mar. '38
Hobbs, W.	Abington Mem., Pa.	Rotating	July '35-July '37
Holmes, J. A.	Mass General, Boston	Pathological	July '35-July '36
Holscher, E. C.	St. Louis City, Mo.	Rotating	July '35-July '36
Howard, G. C.	Hartford Hospital, Conn.	Rotating	July '35-July '37
Jardine, R. R.	R. I. Hosp., Providence	Rotating	July '35-July '37
Jones, J. F.	St. Luke's, Cleveland, O.	Rotating	July '35-July '36
Jorgensen, G. M.	Gorgas, Ancon, Panama.	Rotating	July '35-July '36
Keane, G. P.	N. E. Deaconess, Boston	Pathological	July '35-July '36
Kendrick, M. H.	St. Luke's, N. Y. C.	Surgical	Jan. '36-Jan. '38
Kimball, S.	Peter Bent Brigham, Boston	Medical	June '35-Mar. '37
Kindschi, L. G.	Cleveland City, O.	Rotating	July '35-July '36
Kinney, R.	Bellevue, N. Y. C., 2nd Div.	Medical	July '35-Jan. '38
Kjellesvig, K. L.	Lowell General, Mass.	Rotating	July '35-July '36
Little, M. H.	Bridgeport Hosp., Conn.	Rotating	July '35-July '36
Manning, I. H., Jr.	Boston City, 2nd Service	Medical	Jan. '36-July '37
Martin, A. G.	Lankenau, Philadelphia, Pa.	Rotating	July '35-July '37
McCune, W. S.	Mass. General, Boston	Surgical	July '35-Aug. '37
McKeen, H. R., Jr.	Denver General, Colo.	Rotating	July '35-Jan. '37
Meharg, J. G.	Philadelphia General, Pa.	Rotating	July '35-July '37
Mellen, R. H.	Bellevue, N. Y. C., 3rd Div.	Mixed	July '35-July '36
Miller, C. C.	Palmer Mem., Boston	Surgical	July '35-July '36
Moore, S. H., Jr.	Albany Hosp., N. Y.	Rotating	July '35-July '36
Morgan, W. A.	Springfield Hosp., Mass.	Rotating	July '35-Jan. '37
Mote, J. R.	Mass. General, Boston	Medical	Apr. '36-Nov. '37
Munce, R. T.	Peter Bent Brigham, Boston	Surgical	Feb. '36-July '37
Nesbitt, S.	Fifth Ave., N. Y. C.	Medical	Apr. '36-Apr. '37
Ney, J.	U. of Chicago Clinics, Ill.	Medical	July '35-July '36
Norcross, J. W.	Mass. General, Boston	Medical	July '35-Feb. '37
Noyes, R. E.	Latter Day Saints, Salt Lake City, Utah	Rotating	July '35-July '36
O'Brien, J. V.	Boston City, 1st Service	Surgical	July '35-July '37
Ogden, O. S.	New York, N. Y. C.	Surgical	July '35-July '36
Olcott, C., Jr.	Roosevelt, N. Y. C.	Surgical	Jan '36-Jan. '39
Parsons, H.	St. Luke's, N. Y. C.	Surgical	Jan. '36-Jan. '38
Partington, P. F.	Huntington Mem., Boston	Med. Research	Sept. '35-Sept. '36
Pearman, R. O.	Cleveland City, O.	Rotating	July '35-July '36
Pease, H. B.	Springfield Hosp., Mass.	Rotating	Jan. '36-July '37
Poland, W. M.	Mass. General, Boston	Pediatric	Jan. '36-Oct. '36
Pope, H. G.	Boston City, 2nd Service	Surgical	July '35-July '37
Posey, L. C.	Hillman, Birmingham, Ala.	Rotating	July '35-July '36
Putnam, H. M.	Mass. General, Boston	Medical	Oct. '35-May '37
Rauh, A. E.	Mass. General, Boston	Pediatric	Apr. '36-Jan. '37
Read, B. S., Jr.	Methodist Episcopal, Brooklyn, N. Y.	Rotating	July '35-July '37
Reinhardt, W. I.	Orange Mem., N. J.	Rotating	July '35-July '36

Reynolds, O. S.	Charleston General, W. Va.	Rotating	July '35-July '36
Rhea, R. L., Jr.	Hartford Hosp., Conn.	Rotating	July '35-July '37
Rhodes, P. H.	University, St. Louis., Mo.	Rotating	July '35-July '36
Richardson, R. D.	R. I. Hosp., Providence	Rotating	June '35-June '37
Roberts, C. D.	Boston City, 4th Service	Medical	Oct. '35-Apr. '37
Robertson, J. E.	Peter Bent Brigham, Boston	Medical	Feb. '36-Nov. '37
Rosenow, E. C.	Faulkner, Jamaica Plain, Mass.	Mixed	June '35-June '36
Ross, R. A.	New Haven Hosp., Conn.	Pediatric	Sept. '35-Sept. '36
Ross, R. L., Jr.	Boston City, 5th Service	Surgical	Mar. '36-Mar. '38
Rutherford, F. W.	Children's, Boston	Surgical	Jan. '36-Oct. '37
Saunders, G. A.	Mass. General, Boston	Medical	Apr. '36-Nov. '37
Saxe, I. H.	Beth Israel, Boston	Pathological	July '35-July '36
Shepard, K.			
Sheridan, W. M.	Bellevue, N. Y. C., 1st Div.	Surgical	July '35-July '37
Sherman, R. S.	Memorial, Pawtucket, R. I.	Rotating	Aug. '35-Aug. '36
Shields, J. J.	Germantown, Philadelphia, Pa.	Rotating	July '35-July '37
Simpson, H. N.	Fifth Avenue, N. Y. C.	Medical	Oct. '35-Oct. '36
Simrall, H.	University, Ann Arbor, Mich.	Surgical	July '35-July '37
Snow, R. G.	Alameda Co., Oakland, Calif.	Rotating	July '35-July '36
Snyder, J. C.	Mass. General, Boston	Surgical	Oct. '35-Nov. '37
Soule, G. W.	R. I. Hosp., Providence	Rotating	Feb. '36-Feb. '38
Soutter, L.	Presbyterian, N. Y. C.	Surgical	Feb. '36-Mar. '38
Stafford, D. E.	King Co., Seattle, Wash.	Rotating	July '35-July '37
Strachan, H. L., Jr.	Springfield Hosp., Mass.	Rotating	July '35-Jan. '37
Sunderland, D. A.	Boston City	Pathological	July '35-July '36
Talmadge, S. M.	Mass. General, Boston	Surgical	Apr. '36-May '38
Tanner, D. W.	San Francisco General, Calif.	Rotating	July '35-July '36
Thornley, W. F.	Newark City, N. J.	Rotating	Jan. '36-Jan. '38
Todd, T. C.	Presbyterian, N. Y. C.	Pathological	(Voluntary)
Toole, A. F.	Presbyterian, Philadelphia, Pa.	Rotating	July 35-July '37
Truax, H.	New York, N. Y. C.	Surgical	July '35-July '36
Wallbank, W. L.	Mercer, Trenton, N. J.	Rotating	July '35-July '36
Warner, F. A.	Baltimore City, Md.	Rotating	July '35-July '36
Warner, J. H., Jr.	Mass. General, Boston	Medical	Jan '36-Aug. '37
Watkins, A. L.	Peter Bent Brigham, Boston	Medical	June '35-Mar. '37
Webb, H.	Henry Ford, Detroit, Mich.	Surgical	Sept. '35-Sept. '36
Webster, G. T.	University, Cleveland, O.	Medical	Mar. '36-Sept. '37
Weir, W. C.	Faulkner, Jamaica Plain, Mass.	Rotating	June '35-June '36
West, F. J.	Worcester City, Mass.	Rotating	June '35-July '37
Wheeler, C. A.	Mass. General, Boston	Surgical	Jan. '36-Feb. '38
Whitelaw, G. P.	Mass. General, Boston	Surgical	Oct. '35-Nov. '37
Whitfield, R. D.	Peter Bent Brigham, Boston	Surgical	Oct. '35-Mar. '37
Woolley, P. V., Jr.	Children's, Boston	Bact. & Path.	July '35-July '36
Yeomans, A.	Presbyterian, N. Y. C.	Medical	Feb. '36-Feb. '38

THE NATION'S BIRTH RATE

The report from Washington as published by the Bureau of the Census shows that there were 2,158,919 live births in this country in 1934, a rate of 17.1 per 1,000 population, which is an increase over 1933, when the rate was 16.6.

The three Pacific Coast States had the lowest rate. The high rates were in the Southern States and West Virginia.

The infant mortality for the whole country rose from 58.1 per 1,000 live births in 1933 to 59.9 in 1934.

STATISTICS FOR NEW ENGLAND

	Live Births			Deaths Under 1 Year			Stillbirths		
	Number	Per 1,000 Estimated Population		Number	Per 1,000 Live Births		Number	Per 100 Live Births	
		1934	1933		1934	1933		1934	1933
Connecticut	22,215	13.4	13.6	1,085	48.8	48.4	624	2.8	2.8
Maine	15,719	19.6	18.9	1,112	70.7	66.3	557	3.5	3.7
Massachusetts	63,828	14.7	14.7	3,164	49.6	52.0	1,972	3.1	3.4
New Hampshire	7,869	16.7	15.7	478	60.7	55.9	242	3.1	3.6
Vermont	6,593	18.3	17.0	347	52.6	53.0	217	3.3	3.1

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1934 AND SEVEN YEAR AVERAGE

MONTH ENDING JUNE 22, 1935

	1935					1934			
Diseases	Week ending June 1	Week ending June 8	Week ending June 15	Week ending June 22	Average cases reported for week corresponding to June 22 for past seven years	Week ending June 2	Week ending June 9	Week ending June 16	Week ending June 23
Cerebrospinal Men.	—	1	—	1	—	—	2	2	—
Chicken Pox	94	197	130	101	81	113	145	166	84
Conjunctivitis Inf.	4	5	34	—	—	—	1	—	—
Diphtheria	7	5	2	6	9	—	4	3	6
Dysentery Bacillary	—	—	2	1	—	—	—	1	—
Encephalitis Epid.	—	1	—	—	—	—	—	—	—
German Measles	257	375	323	247	21	6	13	9	6
Influenza	—	3	1	—	—	1	1	—	—
Malaria	—	—	—	—	—	1	—	—	—
Measles	592	761	667	361	173	183	260	210	178
Mumps	62	39	33	23	50	79	64	88	51
Paratyphoid Fever	—	11	5	—	—	—	—	—	1
Pneumonia (Broncho)	18	14	17	12	11	10	7	13	9
Pneumonia (Lobar)	34	28	19	17	15	13	15	6	10
Poliomyelitis	—	—	—	1	—	—	—	—	—
Scarlet Fever	96	64	77	46	35	41	31	41	17
Septic Sore Throat	6	6	5	9	2	4	5	—	4
Trichinosis	—	—	—	—	—	1	1	—	—
Tuberculosis (Pul.)	48	31	35	34	28	29	28	29	21
Tuberculosis (O. F.)	4	1	1	5	3	1	4	3	1
Typhoid Fever	2	2	2	1	1	—	2	1	—
Undulant Fever	1	—	—	—	—	—	—	3	—
Whooping Cough	44	65	62	49	49	28	65	75	52
Gonorrhea	31	33	40	15	37	42	28	82	31
Syphilis	43	43	46	41	66	47	48	54	31

Remarks: No cases of Asiatic cholera, glanders, plague or yellow fever during the past seven years.

CORRESPONDENCE

AN EARLY PLAN FOR PREPAID HOSPITALIZATION

Editor, *New England Journal of Medicine*,

If anyone thinks that a monthly or annual payment to insure hospital care in case of need is a new and novel proposition, it may be of interest to note that on January 1, 1867 Rt. Rev. John J. Power established in Worcester a hospital of a few beds under control of the Sisters of St. Anne's Convent—"to encourage providence and maintain self-respect" a trifling monthly sum was required by which one became entitled to a bed and nursing in time of sickness, or to quote from the announcement "By payment of $3.00 any person may secure the right to a bed with doctor's care, nursing and medicine for one year."

This was the first general hospital in Worcester.

Yours truly,

SAMUEL B. WOODWARD, M.D.

58 Pearl Street, Worcester, Mass.

RECENT DEATH

TURNER—MAURICE WORCESTER TURNER, M.D., aged seventy-eight, of Brookline, Mass., died at his home, 788 Washington Street, June 29, 1935.

Dr. Turner was born in Brooklyn, N. Y., the son of Dr. John Turner and Emma Worcester Turner. He graduated from the Boston University School of Medicine in 1889 and soon after settled in Brookline. He filled the chair of Theory and Practice of Medicine in his Alma Mater and was associated with Dr. Conrad Wesselhoeft in the Department of Pathology and Practice of Medicine. He served on the staff of the Massachusetts Homeopathic Hospital and was a member of the Board of Trustees of the New England Baptist Hospital. In 1911 he was President of the International Hahnemannian Association and held membership in many homeopathic societies.

He was a Mason, Secretary of the Massachusetts Order of Founders and Patriots of America, and at his death was secretary of this body. He was serving

his second year as President of the Sons of the Revolution of Massachusetts at the time of his death.

He is survived by his widow, Mrs. Abby R. (Corliss) Turner, and a daughter, Mrs. Fred B. Maynard, of Arlington.

REPORTS OF MEETINGS

WILLIAM HARVEY SOCIETY

Dr. Jonathan C. Meakins, president of the American College of Physicians, addressed the William Harvey Society at its meeting of April twelfth, at the Beth Israel Hospital. His subject was "Cardiology During the Past Three Hundred Years—The Legacy of William Harvey." Dr. Cadis Phipps presided.

Doctor Meakins briefly reviewed some of the important data in the history of medicine. Michael Servetus, both a theologian and a scientist of the mid sixteenth century, was the first to describe the pulmonary circulation. Many of his conclusions were based on direct observation and were an example of the thought and reasoning of his time. He did not divorce himself from the idea of the spirits of the body which Galen had promulgated. By order of Calvin he was burned at the stake because of his religious theories.

Father Paul may have been acquainted with the valves of the veins, but Fabricius was the first to publish a description of them. William Harvey after fourteen years of study under Fabricius was thoroughly convinced of the circulation of the blood from the right heart to the lungs, and back to the heart where it was pumped into the general circulation. His was the first comprehensive investigation of the circulation and his facts were collected from direct observation and experimentation. He wrote a masterly discussion of this system and gave his proof for his beliefs. Although the book was announced in 1616 it was not published until 1628.

At this time the era of experimentation was in full sway in Great Britain. Bacon was a contemporary of Harvey and insisted on experimental observation as the safe method of investigation. After the publishing of Harvey's book his theories received much opposition and his practice fell off greatly.

Malpighi was born in 1628 and during his lifetime carefully investigated the mysteries of the capillaries. During the next century and a half numerous scientific discoveries were made, and by 1650 many intellectuals had banded together for help, advice, and work. They met weekly in London to discuss philosophical and scientific matters. Among other members were Christopher Wren, architect, and Robert Boyle, physicist.

William Harvey's work was enthusiastically received by the following generation, and further study of the circulation and respiration was carried out. Malpighi with his microscope studied the lung of dogs and demonstrated the small airsacks and their relation to the capillaries which ran between the arteries and the veins. Respiration was thoroughly investigated by several men. The change in the color of the blood as it passed through the lungs was shown to be due to a substance which was absorbed in the lungs. This great wave of investigation swept away much of the supposed knowledge of Galen and Hippocrates. Harvey's legacy consists of the following: first, the overthrow of the authority of the past; secondly, the introduction to the use of hypotheses proved by experimental observation; and thirdly, the use of man and lower animals to obtain experimental data. For some time after the activity of this era men's minds sank into a sort of lethargy, and it was a hundred years after Harvey before there was another important step in the description of the circulation. Stephen Hales, a clergyman, measured the blood pressure and studied the capillaries and their power to dilate and contract. He showed that brandy and certain other drugs could cause a contraction of the capillary vessels and gut, although his observations were not generally accepted for many years. In the eighteenth century Lavoisier demonstrated that the act of respiration removed a part of pure air and gave off carbon dioxide. He showed that nitrogen played a most active part. He suggested that the name oxygen be given to this necessary constituent of air, and he isolated it in pure form. Over a hundred years later Haldane demonstrated the importance of carbon dioxide in the regulation of the respiration.

In recent times there has been a rebirth of experimental observation in medicine. Sir James McKenzie put cardiac disease on a rational clinical basis. His chief work was done before the time of x-ray and the electrocardiogram. In 1896 the sphygmomanometer was introduced clinically.

Doctor Meakins showed several interesting slides to demonstrate his lecture.

AMERICAN MEDICAL EDITORS AND AUTHORS ASSOCIATION

Atlantic City, June 15, 1935.

The sixty-sixth annual convention was called to order by the President, Dr. Dean Lewis, who introduced the Guests of Honor.

Hon. Harry Bacharach, Mayor of Atlantic City, welcomed the membership and presented the Association with a key to the City. The other Guests of Honor, Hon. Edward A. Kenny, U. S. Congress, Hon. Shirley Wynne, M.D., former Commissioner of Health and Hon. S. S. Goldwater, M.D., Commissioner of Hospitals, New York, were next introduced.

Dr. Frederick L. Patry, psychiatrist of the New York State Department of Education, made a plea for the family physician to increase his knowledge of psychiatry in order to handle more scientifically cases of neurosis due to family friction. Doctors A. A. Brill, New York psychoanalyst, and K. Winfield Ney, New York Brain Surgeon, added to Dr. Patry's suggestion by citing many cases and

examples of the results following the proper use of psychiatry in such cases.

Dr. Shirley Wynne told the Association that it was inevitable that a realignment would occur relative to the way medical service would be distributed in the future. At present it is a question whether there will be voluntary or compulsory insurance. State Socialized Medicine looms in the background.

The problems of the Medical Editor were handled by Dr. George Lake, Editor of *Clinical Medicine*. Much enthusiasm was developed concerning his copyright plan to eliminate the throw-a-way journals. He stated that Editors should coöperate with the efforts of the Association in their behalf.

The problems of the Medical Publishers were cleverly handled by Mr. Paul Hoeber. His address gave an answer to many authors as to why contributions which they considered par excellence were not accepted for publication.

After a get-to-gether luncheon, Dr. S. S. Goldwater told the Association of the Hospital Problems he had to contend with. He said that the institutions needed $10,000,000 to continue their work and added that facilities were particularly inadequate for the care of chronic and venereal patients. He said that with the majority of internes receiving but little money from home, owing to the depression, and getting no salaries, they were finding it difficult to continue their training.

Dr. Dean Lewis, President of the Association, addressed the Congress on "Improving the Quality of Medicine." In his address which members will long remember he touched on the subject of fee splitting—a practice he abhorred but would condone if done openly. He said that those who wanted to split fees invariably insisted on secrecy.

Dr. H. Lyons Hunt, Director-General of the Association, closed the program with a report on the financial status and informed the Convention that since Jan. 1, 1935, 220 men were proposed for membership in the Association, of which number 48 had been elected.

At the close of the convention, a resolution was passed putting the Association on record as "basically and fundamentally opposed to State medicine, because it lessens the efficiency of service to the public and the dignity of the profession."

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, JULY 15, 1935

Wednesday, July 17—

†12 M. Clinico-Pathological Conference. Children's Hospital.

Thursday, July 18—

*12 M. Clinico-Pathological Conference. Massachusetts General Hospital.

Saturday, July 20—

*10-12. Staff rounds at the Peter Bent Brigham Hospital. Open to practicing physicians.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

August 29 - September 8—Latin American Congress of Physical Therapy, X-Ray and Radium. For information address Dr. Madge C. L. McGuiness, 1211 Madison Avenue, New York City.

October 7-10—American Public Health Association will meet in Milwaukee, Wisconsin. For information address the American Public Health Association, 50 West 50th Street, New York City.

October 21 - November 2—1935 Graduate Fortnight of the New York Academy of Medicine. See page 898, issue of May 9.

October 28 - November 1—The Twenty-Fifth Clinical Congress of the American College of Surgeons. See page 1065, issue of May 30.

BOOKS RECEIVED FOR REVIEW

Report of the Committee for the Investigation of Sterilization. Boston: American Neurological Association. 132 pp.

The Story of Medicine in the Middle Ages. David Riesman. 402 pp. New York: Paul B. Hoeber, Inc. $5.00.

Précis de Thérapeutique et de Pharmacologie. Septième Édition. A. Richaud et R. Hazard. 1257 pp. Paris: Masson et Cie, Éditeurs. 100 fr.

Medical Practitioners in the Diocese of London. J. Harvey Bloom and R. Rutson James. 98 pp. Cambridge: The University Press. $1.75.

Studies from The Rockefeller Institute for Medical Research. Reprints. Volume 93. 593 pp. New York: The Rockefeller Institute for Medical Research.

The International Medical Annual. A year book of treatment and practitioner's index. 1935. Fifty-Third year. Edited by H. Letheby Tidy and A. Rendle Short. 522 pp. Baltimore: William Wood & Company. $6.00.

Diagnosis and Treatment of Skin Disease. Including the care of the normal skin. Jacob Hyams Swartz and Margaret Gilson Reilly. 316 pp. New York: The Macmillan Company. $3.50.

The Autonomic Nervous System. Anatomy, Physiology, and Surgical Treatment. James C. White. 386 pp. New York: The Macmillan Company. $7.00.

The Diseases of the Endocrine Glands. Hermann Zondek. Third Edition. Translated by Carl Prausnitz. 492 pp. Baltimore: William Wood & Company. $11.00.

Apparatus and Technique for Roentgenography of the Chest. 1935. Charles Weyl and S. Reid Warren, Jr. 166 pp. Springfield and Baltimore: Charles C Thomas.

Child Psychiatry. Leo Kanner. 527 pp. Springfield and Baltimore: Charles C Thomas. $6.00.

The Surgical Clinics of North America. June, 1935. Volume 15, Number 3. Chicago Number. 782 pp. Philadelphia and London: W. B. Saunders Company. Paper, $12.00; Cloth, $16.00 net.

BOOK REVIEWS

Gynecology. Brooke M. Anspach. Fifth Edition. With the assistance of Philip F. Williams and Lewis C. Scheffey. 832 pp. Philadelphia and London: J. B. Lippincott Company. $9.00.

Here are 812 pages of lucid, fluent text obviously written by a scholarly, cultured gentleman. There are 769 excellent illustrations, accurately informative, and conveniently placed. This is especially true of the many diagrams and drawings of operative procedures. The detailed completeness of this book, with its utter lack of verbosity and irrelevancy is most impressive. In addition to gynecology there are five chapters of female urology and two of diseases of the rectum. There is an incomparable sixteen-page discussion of backache perhaps the commonest complaint of women. The turbulent subjects of endocrinology, menstruation, and sterility are presented with erudite conservatism. Consideration of radium and x-ray is adequate for those equipped mentally and instrumentally for their use. Of the main body of the text these portions already mentioned are perfect samples. It is a most excellent book of which the authors should be exceedingly proud—a book well worth purchase and study.

La Renaissance de la Médecine Humorale. Auguste Lumière. 204 pp. Lyon: Imprimerie Léon Sézanne.

This book is by the author of an impressive list of tomes (twenty-three in all) which have dealt largely with a unified theory of disease, and, of course, a simple system of therapy based on that theory. All disease, the theory has it, is based upon a disturbance of the colloids of the body, usually in the nature of a flocculation mechanism, which in turn is dependent upon humoral instability. The author frankly admits the priority of the ancients in this concept and, in his most important chapter (to the reviewer) discusses the various changing views from the times of Hippocrates to the present regarding the importance of the humors in pathological states. He bemoans the fact that the "monumental" work "Le Nouveau Traité de Médecine" comprising twenty-two volumes and written by fifty-two eminent physicians alludes in no single word or phrase to this important theory.

The second chapter of the volume is devoted to an analysis of the reasons behind the abandonment of "humorism" and the necessity for a quick return, because here lies the "key to pathologic and therapeutic enigmas". Lumière's theory is based upon the fact that all bodily cells and the circulating fluids are composed of substances present in colloidal states. Life itself, according to Lumière, is a colloidal set-up which, if disturbed, results in disease; if destroyed, in death. Colloidal flocculation results in the sudden entrance into the bloodstream of insoluble particles and thus in the symptoms of acute disease. These sudden changes are frequently so slight as to pass unnoticed. Certain organs, however, acquire a hypersensitivity to these slight bombardments, i.e., in the bronchopulmonary system, attacks of asthma may develop. With knowledge of these mechanisms, "the mysteries of therapeusis vanish completely".

The process of treating these abnormal humoral states is of the simplest. The author cites the following methods by which the humors may be modified: injections of magnesium hyposulphate, cobalt, colloidal mixtures, and hypertonic solutions; hydration and dehydration, hemolysis, hyperthermia, modification of the pH of the blood, etc. Magnesium hyposulphate in 10 per cent solution given intravenously is recommended most highly. The book is completed by a series of 26 clinical cases illustrating twenty-six therapeutic triumphs and by these words, "Rational and efficacious humoral therapy, of the type we have founded, inaugurates a new medical epoch. . . ." The reviewer sincerely hopes so, but is not quite fully convinced.

Blood Groups and Blood Transfusion. Alexander S. Wiener. 220 pp. Springfield and Baltimore: Charles C Thomas. $4.00.

This book should receive nothing but unqualified approval. It is without doubt the most scholarly and detailed work on the subject of the blood groups in the English language and compares favorably with the German monographs on the subject by Steffan and Schiff. The book may be read with profit by both the beginner and the student who is well versed in the field, since it takes up first principles in great detail and yet delves deeply in the complexities of heredity of the blood groups with the associated subjects of genetics and biometrics. About one-half the book is devoted to individual and species differences in blood groups and much that is new is included, particularly in the sections relating to the agglutinogens M and N of Landsteiner and Levine. The latter workers found, in addition to the four well-known blood groups, that human blood could still further be distinguished by the presence or absence of the "new" agglutinogens M and N. Wiener has done a great deal of investigative work on these interesting factors paying particular attention to their medicolegal applications. Whereas, when the ordinary blood groups are used the chances of establishing paternity of a child, etc., is only about 1 in 6, the addition of test sera containing agglutinogens M and N raises this chance to about 1 in 3. Unfortunately, the preparation of types M and N serum and the interpretation of the reactions obtained with unknown samples of blood are both very difficult and to be entrusted only to an undoubted expert in this very narrow field. Wiener has stimulated much interest in the matter, and it is probably due at least partly to his work that the New York State Legislature recently passed a law requiring blood-grouping tests in all cases of disputed paternity.

The book is exceedingly well printed on good stock

and contains a wealth of references and a good index. There are fifteen pages devoted to the technique of blood transfusion. The writer should be congratulated upon his complete mastery of this rather intricate subject and the book deserves to be made the standard work of reference in the field of blood groups.

Psychology and Health. H. Banister. 256 pp. New York: The Macmillan Company. $2.50.

This is another book by a psychologist on a subject much of which seems to be beyond his ken. Were life and its adjustments all psychology, a psychologist might perhaps adequately handle the topic of psychology and health, but health involves a physical body, subject to traumata, infections, maldevelopments, and malnutritions, and all the adaptive compromises in physical and mental activities subsequent to the temporary or permanent deletions of function which befall an individual.

The author watching the gyrations of the tail forgets too much the dog. He writes well of the inadequacies and fallacies of the theories of Janet, Freud, Jung and Adler. His own theory of sentiment formation is hardly more than a different mode of expressing the thesis that the highest function of the human brain is correlation, which is expressed by an individual in terms of judgment and wisdom.

It is unfortunate that among much that is well said although not new, there appear remarks open to question such as the following: "I do not believe that any breakdown has ever been caused by a study of psycho-pathology although in certain instances a breakdown may have been precipitated. This I consider a good rather than an evil thing, for the earlier the collapse the greater the chance of permanent recovery." With a greater knowledge of psychiatry it is doubtful if the author would be ignorant of the facts that no mental breakdown is so simple as to be attributable to a single factor and that often the earlier a mental breakdown occurs in life the more difficult is the chance of permanent recovery.

Failure of the Circulation. Tinsley R. Harrison. 396 pp. Baltimore: The Williams & Wilkins Company. $4.50.

Harrison divides failure of the circulation into three types—hypokinetic, hyperkinetic and dyskinetic. The first is more commonly known as shock or collapse, the second as effort syndrome, and the third as organic heart disease leading to congestive failure. He discusses these syndromes in twenty-nine chapters and ends with five chapters describing Mixed Types of Circulatory Failure, Failure of the Coronary Circulation and a Summary.

The book is more a work of reference with a review of literature, and a discussion of physiology than a clinical textbook. It is not a book which would be easy reading for one who was not familiar with the modern physiology of the circulation. However, this makes it all the more worthy of careful study, for the material in it frequently provides explanations for conditions in circulatory mechanics which physicians find obscure or which they have been content to dismiss without an adequate analysis. One subject which is well covered is the mechanism of congestive failure and a strong case is made for an explanation of it on the basis of "backward-failure" as described by James Hope 100 years ago, with increase in venous pressure in either greater or lesser circulation, as opposed to the "forward-failure" concept which attributes congestive failure to reduced cardiac output with peripheral anoxemia. Explanations of the genesis of various symptoms and signs such as cardiac hypertrophy and dilatation, dyspnea, Cheyne-Stokes respiration, cardiac asthma, and edema are unusually detailed and the logical presentation of them is very satisfying to one interested in causation. Therapy is less fully considered but the discussion of digitalis is interesting. The author's conclusions that a normal heart rate constitutes tachycardia for the hypertrophied heart, with its difficulty in cellular metabolism of its enlarged fibres, seems well borne out by clinical experience. So too is his decision that "the dilated heart is an inefficient pump for, though it may supply an adequate amount of blood to the tissues, it expends excess energy in order to carry on this work. Heart failure is to be attributed, in the main, to inefficiency rather than to insufficiency of the myocardium."

This book is to be recommended to anyone wishing a summary of the physiology of heart failure in which clinical observations are integrated with an historical survey and experimental data.

Rats, Lice and History. Being a study in Biography, which, after twelve preliminary chapters indispensable for the preparation of the Lay Reader, deals with the life history of Typhus Fever. Hans Zinsser. 301 pp. Boston: Little, Brown & Company. $2.75.

The keynote of this book may be found in the following sentence: "Swords and lances, arrows, machine guns, and even high explosives have had far less power over the fates of the nations than the typhus louse, the plague flea, and the yellow-fever mosquito." The general theme developed by Dr. Zinsser in this most readable account is the rôle that epidemic disease has played in the history of civilization. The earlier chapters cover various of the great epidemics of the past and touch briefly on their effect on contemporary history. On the skeleton of dullest facts he builds, through his literary style, a pleasant flesh which makes an attractive form. Some readers will recognize the first chapter as one that they had already encountered in the pages of the *Atlantic Monthly*, in which Dr. Zinsser pays his respects as an ardent scientist to modern literature.

Clinical and Pathological Applications of Spectrum Analysis. Walther Gerlach and Werner Gerlach. 143 pp. London: Adam Hilger Limited.

This is a very useful, short, practical manual on the clinical and pathological applications of spectrum analysis. The book is essentially a section of "Die Chemische Emissionsspektralanalyse" by Drs. Walther and Werner Gerlach, translated by Joyce Hilger Twyman, and adapted to pathological and forensic problems. Exclusive of the excellent bibliography and index, the text comprises about 130 pages devoted mainly to a description of technical procedures. There are included concise discussions of possible variations in technique and of various pitfalls and precautions to be observed.

After describing various light sources and the operation of the optical apparatus, the book discusses procedures suitable for taking spectrograms of various types of material. These include the analysis (qualitative and quantitative) of organs, secreta, and excreta for the presence of metals and mineral substances. Detailed spectograms are given for gold, silver, copper, bismuth and lead. The application of the method to specific problems of industrial or forensic importance is interestingly illustrated. Among these are the detection of silicon and aluminum in the lung from pneumoconiosis, the finding of mercury in the urine, and the identification of metals in bullet wounds at the site of entrance. There are also useful analysis tables for testing the purity of metals.

The book is tastefully arranged and pleasing in appearance. It should be welcomed especially by workers in toxicology and in the laboratory study of industrial disease. It is particularly valuable to analytical chemists who wish to concentrate upon a highly specialized technique which promises an increasing utility.

What You Should Know about Heart Disease. Harold E. B. Pardee. Second Edition. 127 pp. Philadelphia: Lea & Febiger. $1.50.

This small volume is written for the layman with or without heart disease and is intended to clarify his knowledge of etiology, anatomy, physiology, treatment and prognosis to the end that a better coöperation with the physician may be secured. As is the case with all such books the author has to steer carefully through the sea of controversial matter and in this instance to present what is still unprobed about heart disease only moderately colored with his own point of view. That he is successful in this seems clear and the patient is given enough knowledge and caution to prevent a desire to be his own doctor. In addition to a discussion of causation of heart disease and its manifestations there are chapters on Exercise, Treatment and Climate, and Surgical Risks. Some viewpoints do not conform entirely with those of Boston cardiology. Especially notable is the failure to divide the effects of hypertension on the heart from those of coronary arteriosclerosis as in the statement that "neither diabetes nor high blood-pressure will harm the heart unless the arterial branches supplying blood to the heart muscle are especially affected by arteriosclerosis." Also, lead and alcohol are considered as the most important chemical poisons causing arteriosclerosis. Tobacco is thought to be much less harmful.

While the toxic effects from digitalis are well described, the unpleasant and, at times, alarming symptoms from nitroglycerine are not mentioned. The importance of contraceptive advice for women with heart disease is not considered, and chloroform is advised as an induction anesthetic to precede ether in surgical operations.

This is a book to recommend to certain patients who demand more detail about their disease than can sometimes be discussed in the limited time of an office consultation and will be found helpful in the emphasis of important directions in the conduct of their lives.

Diseases of the Mouth and Their Treatment. A text-book for practitioners and students of medicine and dentistry. Hermann Prinz and Sigmund S. Greenbaum. 602 pp. Philadelphia: Lea & Febiger. $9.00.

This volume is presented as a textbook for practitioners and students of medicine and dentistry with an effort made to treat the entire subject matter as a medico-dental problem. This approach makes the book a welcome addition to stomatological literature. The chapters dealing with the oral manifestations of metabolic disturbances, of blood dyscrasies, of avitaminosis and of the ductless glands; of infectious diseases, tropical diseases, animal parasites and skin diseases, bring together scattered information on this important but neglected subject, and should serve as a convenient and reliable guide for both physician and dentist. These chapters especially will help to clarify some of the many borderline conditions—(The No-Man's Land) which exist between the practice of medicine and dentistry. For this effort alone the book should be highly commended.

The authors have made much freer use of European literature than is common among American writers, and this influence is apparent for many dentists of Europe take the full medical training.

This book with its rather different objective should find its way into the libraries of physicians and dentists interested in modern stomatology.

Stammering and Allied Disorders. C. S. Bluemel. 182 pp. New York: The Macmillan Company. $2.00.

The author believes that "the problem of therapy is simplified if the mechanics of speech are disregarded and if attention is directed to the neurophysiology of speech — one might almost say the biology of speech—in which the production of the

finished word and sentence is regarded as a conditioned reflex. The problem can then be studied in terms of conditioned reflex and inhibition."

There is a rather casual survey of various techniques and theories which have been advanced by others and it seems to the reviewer that the book loses a good deal from this attempt at a fair presentation of utterly irreconcilable points of view, since there is no effective presentation of the author's own methods.

In spite of this rather serious objection there is enough of value in the volume to justify its inclusion in a list of interesting volumes of this baffling problem.

Minor Surgery in General Practice. W. Travis Gibb. 429 pp. New York: Paul B. Hoeber, Inc. $5.00.

Minor Surgery covers a wide and important field. A treatise on the subject is a difficult undertaking. It should be written for the guidance of the general practitioner. It must establish a demarkation between conditions which are within his surgical ability and conditions for which he should seek experienced surgical assistance. The writer of a treatise on minor surgery is therefore confronted with the task of differential diagnosis of minor and major surgical lesions. The general practitioner is fully aware of the possible seriousness of lesions of the thoracic, abdominal and pelvic cavities, but he may err in his evaluation of lesions, especially inflammatory and traumatic lesions, of the head, neck and extremities. The author has in this volume prepared a valuable guide for the general practitioner.

Surgery is a progressive art and happily in recent years has been stirred by great activity. The experienced surgeon in consulting this volume may perhaps be disappointed in failing to find his own method of dealing with a particular lesion. The scope of the volume prohibits the exposition of all methods of treatment. The methods of treatment described are, with very few exceptions, fundamentally sound and safe for the general practitioner. The book, therefore, adequately fulfills its purpose.

The arrangement of text is in part anatomic (regional) and in part pathologic. The introductory chapters are replete with sound advice for the practitioner. The chapter on Shock is elementary but sufficient for a work of this scope. The reviewer would in a subsequent edition like to see included in it specific instructions for the transportation splinting of fractures of the long bones of the extremities. Life-saving expedients, such as tracheotomy, postnasal pack, etc., are well presented.

As a reference book it has a good index. Its examination discloses a number of items in the field of major surgery. Upon consulting the text it is found that these references are to the differential diagnosis of major and minor conditions.

It is a good work. The reviewer hopes that it may be revised in subsequent editions in which certain additions and corrections may be made, e.g., in the administration of antitetanic serum inquiry about previous injections of horse sera and directions for intradermal test for sensitivity; more detailed instruction for the placing of incisions for the drainage of hand infections; warning concerning the thenar branch of the median nerve in opening the thenar bursa, warning against surgery in treating carbuncles of the upper lip; correction of statements regarding suture of tendon sheaths, packing of wounds for bleeding, and removal of one fragment of a fractured carpal scaphoid; and mention of high ligation of the saphenous vein preliminary to the injection treatment in certain cases of varicose veins.

Physically it is octavo, cloth bound, comprises 417 pages, with 148 illustrations, and a double-column index of eleven pages.

Ten Years of Rural Health Work. Rutherford County, Tennessee, 1924-1933. W. Frank Walker. 82 pp. New York: The Commonwealth Fund.

This paper-covered pamphlet of 80 pages describes the "public health activities" for the ten years preceding 1933 in the Kentucky County of which Murfreesboro is the center. The County has an area of about 600 square miles with a population of about 33,000, 25 per cent of whom live under urban or small town conditions. Twenty-five per cent of the total population is colored.

The County had previously been selected by The Commonwealth Fund for a child health demonstration, and a resulting realization of the fact that child health depends on conditions affecting the health of the whole population apparently led to the selection of the county for a general health demonstration.

The work was carried on with the coöperation of The Kentucky State Health Department and of local public-spirited business men, and the Red Cross, as well as of the Commonwealth Fund and the United States Public Health Service.

The schemes for the promotion of public health in Rutherford County consisted chiefly of the customary attempts to supervise personally the lives of individuals at public expense.

If "public health activities" alone justify public appropriations, and if the public benefit of such expenditures is to be appraised by assigning certain arbitrary values to the character and amount of various conventional "activities" then the Rutherford County demonstration with its $1.00 annual per capita cost "scores" a high degree of efficiency. Tangible statistical evidence of public benefit from these "activities" is, however, unimpressive, surprisingly so in view of the large amount of artificial immunization against diphtheria and typhoid fever.

The slightly favorable showing with respect to mortality and morbidity rates for the second five year period as compared with the first, based largely on

estimates of population, is really no more than we might expect to find in any other comparable districts in that part of the country for the same periods. No such districts were selected as "controls" for the purpose of appraisal.

In the annual fluctuations in deaths and in always dubious morbidity statistics, we see the picture of a population in the process of adjusting itself biologically to the unfavorable features of its environment. At the end, as well as at the beginning of the demonstration, the county is still dominated by the same uncontrolled combinations of variable factors affecting life and health. We see no statistical evidence of that abrupt and unmistakable cause and effect relation which may confidently be expected to follow the introduction into a community of a pure water supply, the pasteurization of the milk supply, the connection of human habitations with sewerage systems or even the suppression of flies.

At the end of the demonstration, community and home sanitation in Rutherford County is described as still "on a low plane". In Murfreesboro there was an unsatisfactory water supply and "no adequate" sewerage system. "Domestic sanitation in the rural areas" was "primitive and unsatisfactory". Only about 22 per cent of the milk supply of the population was pasteurized.

It would of course be unwarranted to say that the money spent in this demonstration did no good in the way intended, but the book is chiefly interesting in showing the futility of spending money to promote public health without first assuring the fundamentals of community sanitation.

Sex Practice in Marriage. C. B. S. Evans. 128 pp. New York: Emerson Books, Inc. $1.95.

In this book of one hundred and twenty-eight pages the author confines himself to a discussion of the sex relations of normal married people, and he covers his subject adequately and with good taste. The book contains nothing new or different from the numerous similar books that have been published recently, but it is perhaps a more suitable book to recommend to couples recently married or about to be married because of the omission of much material that would not concern them.

A few statements might be criticized, but they do not affect the purpose of the book.

The Glasgow Royal Maternity and Women's Hospital. Medical Report for the Year 1933. Prepared by D. McK. Hart, Registrar to the Hospital. 184 pp. Glasgow: Aird & Coghill, Ltd.

This somewhat belated volume presents the eighth annual medical report of the Glasgow Royal Maternity and Women's Hospital for the year 1933. This hospital of 175 beds is the largest exclusively obstetric hospital in Scotland. During the year there were admitted 4,595 patients, to whom 3,198 children were born. During the year there were 70 maternal deaths giving an annual death rate of 2.1 per cent. The percentage of stillborn infants, 12.3, seems rather high. There was also a death rate during the hospital period of 4 per cent among the infants born alive. Among the infants born in the Out-Patient Department, there was a stillbirth rate of only 1.3 per cent. The report contains many carefully analyzed statistics which reflect very creditably on the work of the hospital. In general, it does not appear that the morbidity or mortality statistics differ appreciably from those of other first-class obstetric hospitals throughout the world.

Physiology in Modern Medicine. J. J. R. Macleod, and others. Seventh Edition. 1154 pp. St. Louis: The C. V. Mosby Company. $8.50.

It is a great pleasure to see another edition of the outstanding book by J. J. R. Macleod. The recent death of Professor Macleod is indeed a sad loss to medicine all over the world, and it will probably result in making this the last edition of a really great book. This last edition is better than the preceding ones, for the whole book has been improved and brought up to date with a particularly able summary of the modern conceptions of the neuromuscular system by Professor Bard of Johns Hopkins. This outstanding book on physiology certainly ranks among the very best of textbooks. It can be recommended with very great enthusiasm not only to the first-year students of medicine in their course in physiology, but also equally well to any graduate who wants to keep up to date in his knowledge of the physiological mechanisms in the body. It is so written that it should appeal particularly to the clinician, for there are many allusions to disease and the physiological problems involved.

A Textbook of Surgery. John Homans. Third Edition. 1231 pp. Springfield and Baltimore: Charles C Thomas. $8.00.

It is gratifying to see this textbook pass through three editions in five years, for it bespeaks a wide recognition which the book well deserves. This is surgery flavored and seasoned to suit the taste of the most meticulous connoisseur, a book which stamps its author as both eminent artist and teacher.

As in the two previous editions Dr. Homans maintains his lucid and narrative style in adding new information. Authors of textbooks might well take a lesson from the pithiness of his paragraphs. Not only does he give a brief and comprehensive résumé, but his remarks engender curiosity by the very commendable lack of dogma.

In reading the new additions to the present work one is impressed by the impartial acceptance of proved therapy and diagnosis to the exclusion of what still remains obscure or debatable.

To the practitioner Homans makes the old and new facts vibrant and alive; for the teacher he furnishes a sound background for lectures; to the student he has given a delightful text.

Economic Problems of Medicine. A. C. Christie. 242 pp. New York: The Macmillan Company. $2.00.

The author brings together in this book abstracts from the studies of the Committee on the Costs of Medical Care, the report of the Commission on Medical Education, and similar material. His discussion of the data is, on the whole, open-minded and judicial although at times he emphasizes certain aspects of specific matters without a similar consideration of opposing points of view. One feels in some places a defensive attitude with reference to the medical profession. He makes a strong plea for greater interest by physicians in preventive measure and indicates quite clearly the advantages of the extension of private practice into this field.

The reviewer is not entirely sympathetic with his attitude with reference to the place of hospitals in medical practice feeling that he is unduly critical of the extension of work by the hospitals in taking care of the sick.

His final chapter, which discusses a comprehensive plan for medical care, presents clearly and sympathetically various steps in such changes as would seem to be inevitable.

Particularly to be commended throughout his book is the insistence on the need for coöperation between the medical profession and lay individuals and groups interested in improving the quality of medical care. He rightly emphasizes the need of leadership by the medical profession in developing plans which look toward the extension of the benefits of medical science to all groups in the community and the provision of adequate compensation for those rendering the service.

Diseases of the Rectum and Colon and Their Surgical Treatment. J. P. Lockhart-Mummery. Second Edition. 605 pp. Baltimore: William Wood & Company. $10.00.

The first edition of this work appeared eleven years ago and represented a gathering together and revamping of material included in two previous books by the author, one on the rectum and the other on the colon. This present second edition has been largely rewritten and brought quite up to date. The author's long experience as surgeon to St. Mark's Hospital for Cancer, Fistula, and Other Diseases of the Rectum, Etc., in London, gives to his opinions the weight of an unusually extensive acquaintance with the diseases of which he writes. St. Mark's Hospital is outstanding as an institution where a large amount of proctologic work can be seen in a short period of time.

In reviewing this book, one is impressed with the sound common sense shown in the author's discussion of details in diagnosis and treatment. Although due recognition is given to worthy contributions by other writers, the author does not hesitate to emphasize his own ideas when he thinks they are superior. In connection with rectal bleeding, the rôle of an ulcer in a Meckel's diverticulum, as one of the less common causes, is emphasized. Numerous accounts of individual cases of various sorts, many of them unusual and rare, increase the readability of the book. The common and most frequent rectal and anal conditions are systematically covered, each in its own chapter. The ligature operation for hemorrhoids is given preference over other procedures.

The author's operation for correction of complete prolapse of the rectum is given in detail. It is now one of the generally accepted procedures both here and abroad.

In view of the many recent contributions tending to prove almost indisputably that most if not all so-called benign or simple strictures of the rectum are due to lymphogranuloma inguinale, it is surprising that no mention of this fact is made in the chapter on "Simple Stricture of the Rectum."

Lockhart-Mummery has for some years favored the treatment of cancer of the rectum by simple colostomy, followed by perineal excision of the rectum, except in high tumors. His low mortality of 4 per cent in private cases is a good argument for this procedure, and his high five year cure rate is impressive.

This book is complete and well written.

Aids to Surgery. Cecil A. Joll and Reginald C. B. Ledlie. Sixth Edition. 612 pp. Baltimore: William Wood & Company. $2.75.

This very handy pocket edition on surgery is reminiscent of the handbooks on birds and flowers. It is packed full of brief descriptions covering general surgery as well as the specialties, and considers both diagnosis and treatment of the various disease entities. One is truly amazed at what a wealth of accurate information the authors have condensed into 600 pages.

If one has little time for a general review of surgery up-to-date, this volume will give it as briefly as one could ask.

Methods of Treatment. Logan Clendening. Fifth Edition. 879 pp. St. Louis: The C. V. Mosby Company. $10.00.

According to its preface this book "was planned to furnish an *outline* of *all* the methods of treatment in internal medicine". The italics are the reviewer's, because much of the material is presented in the merest outline—and few other methods of treatment than those touched upon can be imagined. In many respects it is up to date—in others it is dogmatically out of date. The illustrations, and much of the text, are primitive for the practicing physician. There are, however, occasional tables and summaries that are entertaining and that under certain circumstances would be very useful. If the reviewer were critically inclined he might prick a hundred holes, but the bottom is so large that still the ship would float. For the medical student it is a comprehensive book; for physicians' reference it is spread too thin; for the library shelves it is handsome, and we are assured by the publishers, durable; for the reviewer it was well worth the two evenings he spent upon it.